THE PATHOLOGY

OF

PULMONARY CONSUMPTION.

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THREE LECTURES

BY

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DISEASES OF THE CHEST, BROMPTON.

ILLUSTRATED BY FIFTEEN WOOD ENGRAVINGS.

HENRY RENSHAW,
356, STRAND, LONDON.
1878.

Ballantyne Press
BALLANTYNE AND HANSON, EDINBURGH
CHANDOS STREET, LONDON

PREFACE.

The following Lectures on the Pathology of Phthisis were delivered at the Hospital for Consumption, Brompton, at the end of last year. Most of the views therein expressed appeared originally in a series of papers in the "Medical Times and Gazette," about four years ago. The wood engravings, drawn by Mr. Collings from my own microscopical preparations, are the same as those used to illustrate my text-book of Pathology and Morbid Anatomy. To my friend and colleague, Dr. J. Mitchell Bruce, I am much indebted for many valuable suggestions in the publication of the Lectures in their present form.

T. Henry Green.

74, Wimpole Street,
April, 1878.

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PATHOLOGY OF PHTHISIS.

LECTURE I.

TUBERCLE AND ACUTE TUBERCULOSIS.

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Gentlemen,—It has for some time past been the custom of the Physicians of this Hospital, set apart for the study and treatment of diseases of the chest, to deliver lectures from time to time on the class of affections which we have here such unusual opportunities of investigating; and it is in accordance with this custom, and by the kind courtesy of my colleagues, that I venture to meet you to-day.

The subject which I have selected for our consideration—the histology and pathology of pulmonary phthisis—is one which, although often before introduced in connexion with others, has never, I believe, been treated separately in this hospital. It is a subject in which we all here must be more or less interested, inasmuch as our knowledge of it is still so far from being complete, and the views held respecting it are so far from being harmonious. It is not my intention to enter into the pathological history of phthisis, and to discuss the various opinions which have been held by different pathologists respecting the nature of the disease. I shall confine myself principally to the consideration of the histological changes met with in the lungs, as illustrated by the microscopical specimens which I shall hope to bring before you, and to the deduction of such pathological conclusions as this histological examination and the present position of our knowledge appear to warrant. I propose to treat my subject under the following heads:—

1. Tubercle and Acute Tuberculosis.
2. The Histological Changes met with in the Lungs in Phthisis.
3. The Pathology of Phthisis.
4. The Varieties of Phthisis.
5. The Influence of Pathology upon the Treatment of Phthisis.

Tubercle and Acute Tuberculosis.

The part which tubercle plays in the production of pulmonary phthisis has long been one of the most vexed questions in pathology. The older views based upon the teaching of Laennec, that phthisis is essentially a tuberculous disease, have undergone various modifications during recent years; and now some, in accordance with the advocacy of the late Professor Niemeyer, would regard tubercle as being only an exceptional element in its causation. These adverse doctrines respecting the tuberculous nature of the disease have obviously, in great measure, resulted from the different senses in which the term "tubercle" has from time to time been employed. In the time of Laennec all caseous products were regarded as tubercular, and phthisis—in which caseation plays so prominent a part—was consequently regarded as a tuberculous disease. When the application of the term "tubercle" became limited by Virchow and his followers to the grey granulation or miliary tubercle, it became evident that in many cases of phthisis at all events no tubercle existed, and that in those cases in which it did exist a large proportion of the consolidation was of such a nature that the term "tubercle" in this limited signification was not strictly applicable to it. Even at the present time much difference of

opinion exists on this head. By some tubercle is still regarded as the most important element in all cases of phthisis; others, again, maintain that it is only an *accidental* accompaniment of the disease; whilst a third class of observers tell us that some cases of phthisis are tubercular, and that others are not. The subdivision of the disease into tubercular phthisis, pneumonic phthisis, and fibroid phthisis, which perhaps represents the tendency of the teaching at the present day, indicates that tubercle is looked upon as, at all events, a very variable element. As in former days, so now, much of this difference of opinion is, I think, to be ascribed to the want of agreement which still exists among pathologists as to what constitutes tubercle. It will be advisable, therefore, before proceeding to consider the pulmonary lesions met with in phthisis, to endeavour to answer this question.

Whatever views may be entertained by different observers respecting the nature of tubercle, I presume that all will admit that it is met with in its most typical form in the disease known as acute miliary tuberculosis. To whatever other morbid products we may apply the term "tubercle," the small disseminated growths met with in the lungs and in other organs in this disease have certainly the first claim to be thus designated. Let us, then, examine in the first place the lesions met with in

the lungs in acute miliary tuberculosis. We will confine ourselves to the lungs, because it is with these organs that we are now especially interested. The consideration of this subject will not only help us to define tubercle, but it will contribute very materially towards the proper understanding of phthisis itself.

When the lungs of a person who has died from acute miliary tuberculosis are examined, they are found studded more or less uniformly throughout with small nodular growths which are universally known as miliary tubercles. These growths are of two kinds—the *grey* and the *yellow*. The grey are semi-transparent nodules of a greyish-white colour, varying in size from a small pin's head to a hemp-seed. They are somewhat spherical in shape, and usually possess a well-defined outline. Sometimes they are firm and almost cartilaginous in consistence, whilst in other cases they are much softer and almost gelatinous. These softer forms, instead of being semi-transparent, are more opaque and white. The yellow are, for the most part, larger than the preceding, many of them much so; some being as large or larger than a pea. They are also softer in consistence, less defined and regular in outline, and they pass more insensibly into the surrounding tissue. Many of them possess a greyish-white translucent margin, which may be

pretty firm in consistencc, but is never so hard as are many of the grey nodules, whilst their central portions are opaque, yellowish, or caseous.

So much for the general naked-eye characters of the growths. There are, however, certain points connected with the manner in which they are associated, and with the condition of the surrounding pulmonary tissue, which it is important to bear in mind, as they help us to understand the pathology of the disease. Firstly, with regard to the manner in which the two kinds of growth are associated. Both kinds are frequently found in the same lung, some of the nodules being small and grey, others larger and of a yellowish colour. It is, however, more common to find nothing but the smaller grey nodules, the yellow growths being entirely absent. In a less frequent class of cases nearly all the growths are of the yellow variety. Secondly, with regard to the condition of the surrounding pulmonary tissue. This may be normal, more or less congested and œdematous, or it may present varying sized tracts of granular friable consolidation, which exhibit all the physical characters of hepatised lung. The lungs generally are large, and their edges emphysematous. A perfectly normal condition of the intervening pulmonary tissue is found in many of those cases in which the growths are of the firm grey variety; but with the yellow

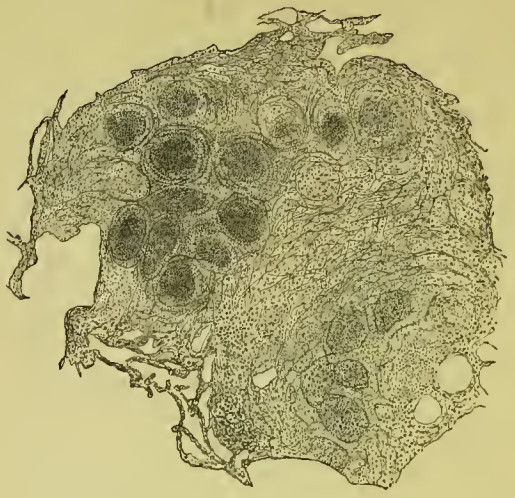
and soft grey nodules, the lungs are nearly alway more or less congested or consolidated.

We proceed, in the next place, to consider the constitution of these tuberculous growths. Their microscopical, like their macroscopical characters, present some differences. It may be stated generally that they consist of three kinds of structure :— 1, Cellular elements accumulated within the alveoli, such as are met with in the so-called broncho- or catarrhal-pneumonia ; 2, A cellular infiltration and thickening of the alveolar walls ; and 3, A reticulated tissue associated with large multinucleated branched cells. These three kinds of structure are variously combined, in some cases the nodules consisting mainly of one kind, in others of another. These differences in the constitution of the growths appear to depend, as will be seen presently, upon differences in the *stage* of their development, and in the *intensity* of the tuberculous process. The histological variations met with in the growths are rarely associated with any corresponding well-marked variations in macroscopical characters. The nodules in the earlier stages of their development, however, are for the most part less sharply defined, softer, and wanting in that firmness of consistence so common in the older tubercles.

In describing the histology of the growths it will be well to begin with those which appear to be in

the earlier stages of their development. Cases of acute tuberculosis are occasionally met with in which most of the pulmonary nodules consist in the main of products accumulated *within* the alveoli (Fig. 1). Each nodule corresponds with a group of alveoli

Fig. 1.



A small soft Grey Tubercle from the Lung in a case of Acute Tuberculosis. The whole of the tubercle is shown in the drawing, and it is obviously constituted largely of *intra*-alveolar products. × 100, reduced to ⅓.

containing cellular elements, which are often associated with an amorphous or faintly granular material. The cells are for the most part large, granular, and nucleated. Many of them are evidently the detached and swollen epithelium of the

alveolar walls, whilst others must be regarded as the products of epithelial proliferation. In addition to these, there are some smaller cells resembling leucocytes, which are probably in the main emigrants (Fig. 2). With regard to the source of the amorphous or faintly granular material, it is difficult to speak with certainty. I am, however,

Fig. 2.

A portion of a small soft Grey Tubercle from the Lung. This is from a case of acute tuberculosis, probably in an earlier stage than that from which Fig. 1 was drawn. The figure shows one of the alveoli filled with epithelial elements and a few small cells, with some cellular infiltration of the alveolar wall. × 200.

inclined to believe that much of it is the product of some retrograde metamorphosis, probably mucoid, of the epithelium, although possibly it may be to some extent an exudation from the blood-vessels. I have never seen it present anything like the appearance of the fibrinous exudation of a croupous pneumonia. The tuberculous nodules are occa-

sionally constituted almost entirely of these *intra*-alveolar products, the alveolar walls being but little if at all altered. This slight or non-implication of the alveolar walls is, however, but rarely met with, probably, I believe, because if not involved from the first, they tend in all cases to become so very early in the process.

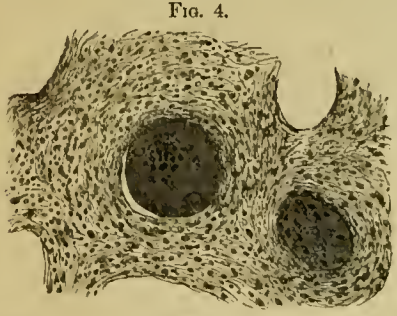
Much more common than the preceding are cases in which, associated with the accumulations within the alveoli, there is a marked cellular infiltration of the alveolar walls (Fig. 3). The alveolar septa are infiltrated with small, round (lymphoid) cells, and are thus more or less thickened. This infiltration and thickening of the alveoli is exceedingly constant, and, as I have just indicated, is probably wanting only in the earliest stage of some exceptional cases. To this I will allude again presently. It is by no means uncommon to meet with cases of tuberculosis in which most of the pulmonary nodules consist solely of the intra-alveolar accumulations and alveolar infiltration just described, and in which the reticulum and giant cells, now so often regarded as essential to the constitution of tubercle, are completely absent. Upon this I would venture especially to insist.

In cases of acute tuberculosis in which the process is, presumably, in a more advanced stage, the structure of the pulmonary nodules differs somewhat from the preceding. The thickening of the

Fig. 3.

A portion of a Yellow Tubercle from the Lung in a case of Acute Tuberculosis. Showing the degeneration of the central portions of the nodule *c*, and the cellular infiltration of the alveolar walls and accumulations within the alveolar cavities at the periphery *p*. × 100.

Fig. 4.



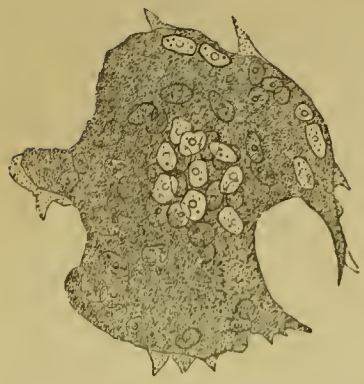
A portion of the more external part of a Grey Tubercle from the Lung in a case of Acute Tuberculosis. Showing the extensive infiltration and thickening of the alveolar walls, and the giant cells within the alveolar cavities. × 100.

alveolar walls is more marked, and some of the alveolar cavities contain large multinucleated masses of protoplasm—the so-called "giant cells" (Fig. 4). Only two or three of these cells may be found in a single nodule, and, as stated by Dr. Klein, they usually appear first in the more peripheral portions.* The alveoli which are not thus occupied contain the smaller cellular elements and amorphous matter already described.

I must now allude somewhat more fully to the giant cells, because they appear to play a very prominent part in the further development of the tuberculous nodule. The characters of these cells and the changes which they undergo have been described in detail by E. Wagner, Schüppel, and Buhl, with whose descriptions I, in the main, agree. The cells, as first observable, are large masses of faintly granular protoplasm, of a spheroidal shape and somewhat irregular outline, containing numerous round or roundly oval nuclei with one, and sometimes two, bright nucleoli. They vary in size; the larger may measure as much as $\frac{1}{200}$ of an inch in diameter. The nuclei may be exceedingly numerous, as many as thirty being occasionally met with in a single cell. Most of them are usually either collected together about the central part of the cell, or distributed principally at its periphery (Figs. 5 and 6).

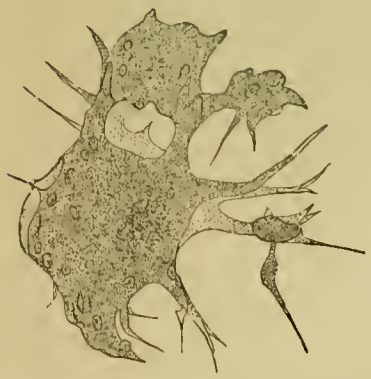
* "The Anatomy of the Lymphatic System," II. By E. Klein, M.D. Page 74.

Fig. 5.



A Multinucleated Cell from the Lung in a case of Chronic Phthisis. Showing the large number of nuclei with bright nucleoli. × 400.

Fig. 6.



A Multinucleated Cell from the Lung in a case of Chronic Phthisis. Showing the long branched processes, which are continuous with the reticulum of the surrounding indurated growth. Some of the processes are in connexion with smaller nucleated elements. × 200.

With regard to the origin of these giant cells in the lung—the statement of Dr. Klein* that they originate either by the fusion of the alveolar epithelium or by the excessive development of a single epithelial cell, appears to me to be probably correct. They are certainly situated distinctly within the alveolar cavities.

With the further development of the tubercle the number of giant cells increases, so that most of the alveoli may be thus occupied. These cells now play a prominent part in the process. They increase in size and send out long branched processes, from which are often developed smaller protoplasmic masses (see Fig. 6, p. 13); and in this way a branched reticulum is produced around the original giant cell, within the meshes of which are sometimes seen a few unaltered or enlarged epithelial elements. Coincidently with these changes in the giant cells the small lymphoid cells infiltrating the alveolar walls, already alluded to, become developed into an imperfect fibro-nucleated structure, which in many parts may closely resemble adenoid tissue. This new tissue, which contains no new blood-vessels, is in direct histological continuity with the branched reticulum developed from the giant cell (Fig. 7). The fully developed tuberculous nodule thus con-

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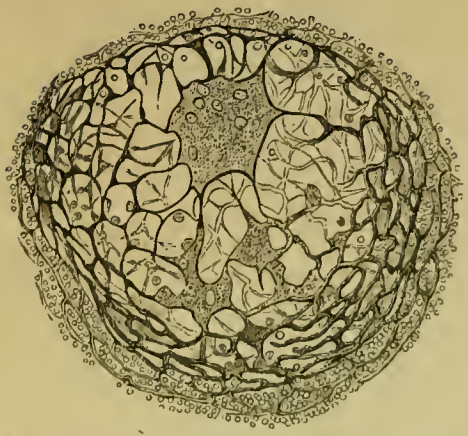
* *Loc. cit.*

Fig. 7.



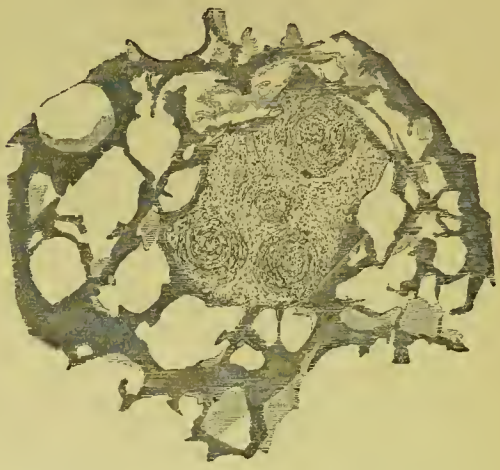
Multinucleated and Branched Cells from a firm Grey Miliary Tubercle of the Lung in a case of Acute Tuberculosis. Wide meshes are seen in the immediate vicinity of the cells enclosing a few lymphoid elements. The branched processes are directly continuous with the adenoid reticulum of the tubercle. × 200.

sists of several giant-cell systems, each of which is surrounded by a fibro-nucleated tissue (Fig. 8).

We must now consider the secondary changes which take place in the tubercle. The nodules invariably undergo more or less retrograde metamorphosis, although the extent of this varies considerably in different cases. The retrograde change commences in the centre of the nodule, and it proceeds most rapidly and is most extensive in those nodules which consist mainly of intra-alveolar products, and in which there is but slight infiltration of

the alveolar walls and a complete absence of the giant-cell reticulum. (See Fig. 3.) Upon this disintegra-

Fig. 8.



A firm Grey Tubercle from the Lung in a case of Acute Tuberculosis. Showing the grouping of the elements around separate centres, the nodule consisting of several giant-cell systems. × 33.

tion of the nodule in the earlier stages of the process, *before* the development of the reticulum, I would especially insist. Very many of the larger, softer, grey, and yellow tubercles are nodules which have thus undergone retrograde change in this early stage.

In other cases the retrograde metamorphosis does not occur until much later—after the development

of the giant-cell reticulum. The degeneration then usually takes place much less rapidly, and is much less complete, although its extent varies in different cases. The fibro-nucleated structure at the periphery of the nodule rarely undergoes complete degeneration, but tends to form an imperfect fibroid tissue around the central more degenerated parts. The giant cells sometimes undergo a peculiar fibroid transformation prior to their degeneration, becoming converted, according to Dr. Klein,* into a dense fibrillar substance. It is these less degenerated and more fibroid nodules which constitute most of the firmer small grey tubercles of the lung.

With regard to the cause of the retrograde change—it is undoubtedly mainly due to imperfect vascular supply. It is well known that tubercle in the later stages of its development is non-vascular, and that in artificially injected preparations the injection does not permeate the tuberculous nodule. It is stated by Dr. Klein that in the earliest stages of the process, when the nodule consists almost exclusively of intra-alveolar products, the pulmonary capillaries are impermeable, and that this impermeability is mainly due to the pressure exercised by these products upon the

* *Loc. cit.*

alveolar walls. But whilst admitting the influence of intra-alveolar pressure, there can, I think, be little doubt that it is the cellular infiltration of the alveolar walls which is the most important element in the causation of the vascular impediment; the cellular infiltration leading to the occlusion of the pulmonary capillaries.* Although the retrograde metamorphosis of tubercle is thus, in great measure, due to the obliteration of the blood-vessels in the process of its growth, the inherent low vitality of the new elements must also be regarded as an important causative factor. To the existence of this is probably to be partly ascribed not only the absence of any new formation of blood-vessels in the fibro-nucleated tissue, but also the production of the giant cells. To this I shall have occasion to allude again presently.

In passing from the histology of the pulmonary tubercles to more pathological considerations, it is not my intention to enter at all fully into the pathology of acute tuberculosis. I shall assume it to be an infective disease, the tubercles being in-

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* Rindfleisch describes an extensive proliferation of the endothelium of the blood-vessels, and regards this as an important cause of the bloodlessness of the nodules. Ziemssen's "*Cyclopædia of the Practice of Medicine*," vol. v. p. 645.

flammatory growths, resulting from the dissemination of infective particles by means of the blood-vessels and lymphatics from some inflammatory infective focus—a focus which, in the majority of cases, although by no means invariably, is the product of a scrofulous inflammation. The correctness of this view, first promulgated by Buhl, I presume now to be well established. What I am particularly desirous of bringing somewhat prominently under notice are what appear to me to be the causes of the differences which we have seen to exist in the constitution of the tuberculous nodules, and the relation which subsists between these differences and those met with in other inflammatory lesions. In doing so I must allude, in the first place, to an opinion which has been held by some, that a pathological distinction must be made between the intra-alveolar accumulations and the small-celled growth in the alveolar walls. The former have been supposed to occupy a different pathological position to the latter—they have been looked upon as *pneumonic*, whilst the latter only have been regarded as *tuberculous*. In the present position of our knowledge of the histology and pathology of pulmonary tuberculosis, it appears to me that any such view of the relation between these intra- and inter-alveolar changes is altogether untenable. The intra-alveolar accumulations and

the alveolar growth do not stand to one another in the relation of cause to effect, but are both the direct result of one common cause—the irritant derived from the focus of infection.

With regard to the causes of the differences which exist in the constitution of the tubercular nodules, it has already been intimated in the description of their histology that they are in part due to their *age* —the stage of their development. I stated that in exceptional cases of acute tuberculosis—cases in which presumably death occurred in the earliest stages of the process—the pulmonary nodules consisted principally of intra-alveolar products; but that in the more common cases, where death occurs later, a small-celled growth in the alveolar walls, and the presence of giant cells, constituted the predominant structural change. But whilst admitting the importance of the element of time as a cause of these histological differences, I must express my belief that they are partly due to *differences in the intensity of the tuberculous process.* My grounds for this belief are based partly on analogy, and partly on the histology of the lesions. It must be admitted that the intensity of an inflammatory process, whether primary or infective, determines to a great extent the nature of the resulting textural changes. Intensity, it must be borne in mind, comprises two factors—one, the most important,

the severity of the injury to which the inflammation owes its origin; the other, less important, the susceptibility of the injured tissue. In inflammations of considerable intensity—the so-called acute inflammations—exudation, emigration, and in the case of epithelial and endothelial structures, cell-proliferation constitute the principal structural changes; whereas, in inflammations of less intensity and longer duration—the chronic inflammations, the vascular phenomena are less pronounced, and the principal change consists in the development of a fibro-nucleated tissue around the blood-vessels. Applying this general law to the case of tuberculous lesions in the lung, we should expect that the greater the intensity of the tuberculous process the more prominent would be exudation and epithelial proliferation; the less its intensity, the greater the tendency to the development of a fibro-nucleated tissue in the alveolar walls.

Turning now from analogy to what is actually observed in the lungs, two of the facts connected with the histology of the disease which I have already brought before your notice appear to me to go very far towards proving the important position which the degree of intensity of the tubercular process occupies in determining the constitution of the pulmonary lesions. Of these, one is the softening and disintegration of the nodules in the earlier

stages of their development, when they consist mainly of products accumulated within the alveoli, and the completeness of the retrograde change in nodules thus constituted. That cases of acute tuberculosis are not unfrequently met with in which complete disintegration of the nodules takes place before there is any considerable alveolar thickening, and before the development of the giant-cell reticulum, whilst in other cases the retrograde metamorphosis is less complete and does not occur until the last-named changes have taken place, appears to me to prove conclusively that something more than the element of *time* is required to account for the difference.

The other histological fact which tends to support the position I am attempting to maintain, relates to the condition of the pulmonary tissue between the tuberculous nodules. I stated that in those cases of acute tuberculosis in which the pulmonary nodules are firm and grey—those cases, that is to say, in which development has proceeded to the formation of the giant-cell reticulum, the intervening pulmonary tissue is often found perfectly normal; whereas in those cases in which the nodules are opaque and soft, or yellow, consisting largely of intra-alveolar products, this tissue is always more or less consolidated; the consolidation being due partly to hyperæmia, and partly to swelling and

probably proliferation of the alveolar epithelium. These differences in the condition of the intervening pulmonary tissue are undoubtedly partly owing to differences in the duration of the tuberculous process, as after the lapse of a sufficient length of time much of the intervening pulmonary consolidation may become absorbed. They are, however, to be partly accounted for, I believe, by differences in the intensity of the tuberculous process; when this is considerable, the irritative influence being exercised over wider areas and so causing more diffused lesions.

From these considerations therefore I conclude that, although the differences met with in the constitution of the pulmonary lesions in acute tuberculosis are partly due to differences in their *age*, they are also influenced to a large extent by differences in the *intensity* of the tuberculous process, and I would submit—

1st. A tuberculous process of considerable intensity tends to give rise to lesions in which epithelial proliferation, and sometimes exudation, occupy a prominent place, and the lesions are prone to undergo early and rapid retrogressive changes—before the development of the giant-cell reticulum.

2nd. In a process of slight intensity epithelial proliferation is but little pronounced, and probably in some cases completely absent; the development

of the giant-cell reticulum and of the fibro-nucleated tissue in the alvcolar walls reaches its maximum, and whilst slight rctrograde changes occur in the central, in the peripheral portions of the nodule there is a tendency to fibroid development. In submitting these propositions, I would again repeat that the intensity of the process depends not only upon the severity of the infective agent, but also upon the susceptibility of the pulmonary tissues.

Thus far I have confined myself to an attempt to account for the differences met with in thc constitution of pulmonary tubercles, and I have endeavoured to establish the existence of an analogy between these differencces and those common to all inflammatory lesions. The most important point, however, still remains for our consideration—viz., the relation which subsists between these tuberculous growths and the products of non-tuberculous inflammations. In order to answcr this question, I must ask you to consider certain well-known facts connected with the pathology of *scrofula*.

The constitutional condition known as scrofula—a condition usually inherited, but sometimes acquired—is characterised, as you are aware, by certain pathological tendencics. Of these, the most important, and that which especially concerns us in our prcscnt inquiry, is a great susceptibility of certain tissues to injury, and a pcculiarity in the

course and in the products of the inflammation which the injury induces. This susceptibility, more or less general, is usually most marked in the mucous membranes and in the lymphatic glands, although the part which is the most prone to suffer varies considerably in different cases. Not only is there this susceptibility to inflammation, but the inflammatory process tends to be exceedingly protracted, it is very readily reinduced, and the alterations produced in the part differ from those caused by inflammation in a healthy person. When inflammation occurs in a healthy individual, if it does not cause the death of the part, the inflammatory products either become absorbed, or the process leads to suppuration or to the formation of a vascular fibro-nucleated tissue. In scrofulous inflammation, the absorption of the inflammatory products is very much less readily effected; they tend to infiltrate and accumulate in the tissue, where by their pressure they interfere with the circulation and so lead to retrograde and caseous changes. There is but little or no tendency to the development of new blood-vessels, and hence there is no organisation of the new growth.

These peculiarities of inflammation in scrofulous subjects are to be in great measure ascribed to that inherent low vitality of the tissues which obtains in this disease, and also to certain peculiarities in

the histology of the inflammatory products. Virchow long ago pointed out the richly cellular character of the products of scrofulous inflammations, the tendency of the cells to infiltrate the tissue, and the extreme tardiness with which the infiltration becomes absorbed. Quite recently Professor Rindfleisch has stated that these cells are for the most part *larger* than those met with in normal inflammation; and this being the case, their removal by passage into the lymphatics is less readily effected.* This largeness of the cells is well shown in the specimen from which this drawing was made (Fig. 9). During the present year Mr. Godlee has called attention to the existence of very large cells in the products of scrofulous inflammation of joints.† In tubercle, the close relation of which to scrofulous lesions we are now considering, the existence of large cell-forms—not only of the so-called giant cells, but of large cells of an epithelial type—has been established by Buhl, Wagner, Schüppel, and other observers.‡ When speaking of the giant cells in pulmonary tubercle, I indicated that their

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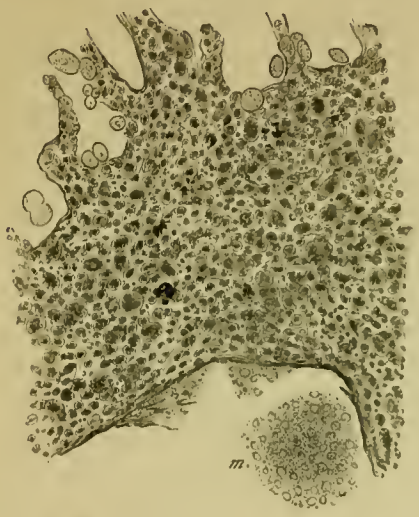
* Ziemssen's "Cyclopædia," *loc. cit.*

† "Trans. Path. Soc. Lond.," vol. xxviii. p. 449.

‡ These cells of epithelial type appear to occupy an intermediate position between the giant cells and lymphoid elements. They are best seen in tuberculosis of lymphatic glands, and are, I believe, much less commonly met with in the lung.

origin was to be partly ascribed to the low vitality of the inflammatory products, and respecting the pathological significance of these large multinu-

Fig. 9.



Scrofulous Inflammation of a Bronchus. Section of a small bronchus of a markedly scrofulous child, the subject of bronchitis, which terminated in miliary tuberculosis. The deeper structures of the bronchial wall are seen to be extensively infiltrated with cells, most of which are *larger* than those met with in the less extensive infiltration of healthy inflammation. The infiltration extends to and invades the walls of the adjacent alveoli, which are seen at the upper part of the drawing. The cavity of the bronchus contains a little mucus, *m*. × 200, reduced ½.

cleated masses of protoplasm, both here and in other situations, I would again express the opinion which I ventured to do elsewhere some three years ago—

that they are the result of an inflammatory process of slight intensity, occurring in tissues of such low vitality that the cellular inflammatory products are incapable of forming an organised tissue, but merely undergo some increase in size and then tend to slowly degenerate. The protoplasm grows, the nuclei multiply, but that higher manifestation of vitality—the division of the cell—does not take place.

These histological peculiarities of the products of scrofulous inflammations not only lead to an extensive and obstinate infiltration of the affected tissues, but, as insisted upon by Professor Rindfleisch, they must, in the parenchyma of organs, as in the glands and viscera, cause, by the pressure they exercise, more or less obstruction of the blood-vessels, and so interfere with the vascular supply. To this interference with the vascular supply, to which I shall have occasion to allude more fully when speaking of phthisis, is to be mainly ascribed the retrograde and caseous changes which are so characteristic of scrofulous lesions.

Having thus indicated some of the more important points in the pathology of scrofula, it remains to consider the relation which subsists between scrofulous inflammation and the disseminated inflammatory lesions met with in the lungs and other organs in acute miliary tuberculosis. The close

analogy between the two is obvious. There is in both the same tendency to cellular infiltration (which we have seen exemplified in the lungs by the infiltration of the alveolar walls), the same development of large cells, the same vascular obliteration and retrograde change. It is well known that acute tuberculosis is especially liable to supervene in scrofulous persons, and inasmuch as the tubercular process is due to the dissemination of infective particles from some focus of inflammation, the tubercular lesion or product of the secondary, naturally tends to resemble in its histological characters the primary scrofulous inflammation.

Acute miliary tuberculosis, however, does not occur exclusively in those who are scrofulous; the primary inflammation does not in all cases possess the scrofulous characters I have enumerated. It must be admitted that the products of a non-scrofulous inflammation may under certain circumstances become infective, and give rise to a general or more local tuberculosis. Of the nature of these circumstances we are at present ignorant. They may possibly be connected with atmospheric influences or with certain undefined conditions of the organism. The general tuberculosis, however, whatever be the nature of its inflammatory antecedents, presents but little histological diversity. The lesions are in all cases characterised by their bloodlessness and ten-

deney to retrograde change, although this tendency is certainly more marked in those cases in which there is a marked scrofulous constitution. It is, for the most part, in those who are distinctly scrofulous that disintegration is the most rapid and extensive; whilst in those who are not scrofulous the firmer and more fibroid tubercles are especially met with. These differences again illustrate what I have stated respecting the influence of the intensity of the process upon the histology of tubercle.

In concluding this part of my subject, the question naturally arises—what, then, constitutes tubercle? From a consideration of the histological characters of the miliary lesions met with in the lungs in acute miliary tuberculosis, it seems to me difficult to frame a definition of pulmonary tubercle upon a purely histological basis. The formation of giant cells and of a giant-cell reticulum is undoubtedly the most characteristic change; but then it occurs only at a certain stage of the process, and it would, I think, be illogical to exclude lesions from the category of tubercle simply on the ground that this giant-cell formation is absent. From the close histological relation which subsists between the disseminated nodules met with in acute miliary tuberculosis and the more diffuse lesions produced by primary inflammation in scrofulous subjects, the inflammatory products in both

cases might be described as *tuberculous*. Such an extension of the significance of tubercle would, I think, be convenient. But restricting the use of the term to the disseminated miliary lesions so characteristic of an infective process, tubercle may, I think, be defined to be the product of an inflammatory process of slight intensity, and confined to a small circumscribed area of tissue, the cellular elements of which, owing probably to some constitutional weakness, are of such low vitality that they are not only incapable of forming an organised tissue, but from their difficulty of removal and consequent tendency to accumulate, interfere so much with the circulation as to lead to more or less retrograde metamorphosis of the affected area.

LECTURE II.

THE HISTOLOGY OF PHTHISIS.

Definition of Phthisis—Histological changes met with in the lungs—Fibrinous exudation and leucocytes within the alveoli—Epithelial accumulation—Macroscopical characters of lungs—Cellular infiltration of the alveolar walls—An increase in the interlobular connective tissue—Changes in the bronchi—Ulceration of bronchial mucous membrane—Alterations in the adjacent pulmonary tissue—Pathology of Phthisis—Nature of morbid processes—Old doctrines—Tubercular nature of the disease—Relation of phthisical to other pulmonary lesions—Inflammatory nature of phthisical consolidation of the lung—Variations in the lesions due to differences in the intensity of the inflammatory process.

Gentlemen,—Last time we were occupied with the consideration of the pulmonary lesions met with in acute miliary tuberculosis. I attempted to account for the histological differences in these lesions, to point out the relation which subsists between them and those caused by non-tuberculous inflammation, and to answer the question—what constitutes pulmonary tubercle? We are now in a position to study pulmonary phthisis. Before doing so, however, it is important to understand clearly what forms of lung consolidation are to be included

under this head, in other words, to give a rough anatomical definition of the disease. There is perhaps rather a tendency in the present day, when attempts are being made to subdivide phthisis into distinct pathological varieties, to extend the signification of the term, and to include all forms of pulmonary disease which run a more or less chronic course, and which are attended by cough, expectoration, and emaciation, within the phthisical category. There are many such morbid conditions of the lung, which differ so materially both in their pathological history and clinical phenomena from that large class of diseases usually included under the common term of "phthisis," that it would appear to me convenient to exclude them from the category. Amongst the most important of such conditions I would name chronic pneumonia, including Corrigan's cirrhosis, the inflammatory disorganisation of the lung which results from pressure on a bronchus, and pulmonary lesions which are distinctly syphilitic. The pathology of these conditions it is not my intention in the present lectures to consider. I shall confine myself to those forms of lung disease which come more properly within the category of phthisis. These forms of lung disease are all characterised by the progressive character of the pulmonary consolidation, and by the subsequent softening and disintegration of much of the conso-

lidated tissue; and also by the fact that the upper portions of the lung are in almost all cases the first to become involved. The progressive character of the consolidation and the subsequent disintegration which it undergoes must, I think, be regarded as the most distinguishing features. They are in themselves sufficient to separate phthisical from the other forms of lung consolidation I have enumerated. Phthisis as thus anatomically defined comprises a class of affections which present most diverse clinical phenomena, and it is to the histology and pathology of these affections that I would now direct your attention.

If the lungs in the various forms of pulmonary phthisis be examined microscopically, it will be found that the histological changes which have taken place in them are mainly of four kinds:—1st. The presence within the pulmonary alveoli of a fibrinous exudation and leucocytes; 2nd. An accumulation of large epithelial cells within the alveoli; 3rd. An infiltration and thickening of the alveolar walls with small cells, together with, in most cases, a similar change in the walls of the terminal bronchioles; and 4th. An increase in the interlobular connective tissue. These four kinds of morbid change are very constantly associated, although in very different degrees, and some are more prominent and characteristic than others. Upon the prepon-

derance of one or other of them mainly depend those variations in the physical characters of the lungs which are met with in the different stages, and in the different varieties of the disease.

I propose, in the first place, to describe somewhat minutely these various kinds of morbid lesion, and to point out the more important alterations which they respectively produce in the physical characters of the lungs. Their pathological significance, together with the relation in which they stand to one another and to other pulmonary lesions, we will consider subsequently.

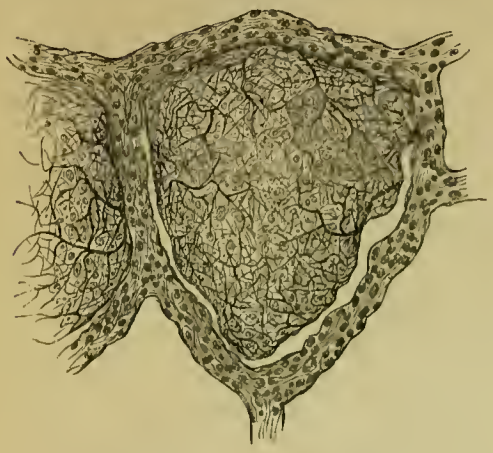
It may be stated generally that the changes met with in the lungs in pulmonary phthisis are similar histologically to those which occur in acute miliary tuberculosis. They differ, however, especially in two particulars. In the first place, whereas the lesions in acute tuberculosis, owing to the infective character of the disease, tend to be limited to small circumscribed areas, those of phthisis more commonly involve wider and more diffused tracts of tissue. Secondly, the element of time impresses certain peculiarities upon the lesions in a large proportion of the cases of phthisis; for inasmuch as this disease usually runs a much more protracted course than acute tuberculosis, the lesions not only become more densely fibroid, but they are also frequently the seats of secondary changes. It must

also be borne in mind that in a large number of cases phthisis is accompanied by a local tuberculosis, which gives rise to lesions in all respects precisely similar to those which have been already described as resulting from the more general disease.

1. *The Presence of a Fibrinous Exudation and Leucocytes within the Pulmonary Alveoli.*—This, although less frequently met with than the other forms of lesion, often contributes largely to the production of phthisical consolidation of the lung. In making this statement, I am aware that I differ somewhat from some other pathologists; but I have observed the change so frequently that I must venture to express the opinion that it by no means infrequently constitutes an important element in the disease. The exudation material presents a perfectly characteristic appearance, and cannot be confounded with the mucoid substance which is so frequently seen in the air-vesicles in cases of so-called broncho-pneumonia. It is precisely similar to that which fills the alveoli in acute croupous pneumonia, consisting of an obscurely fibrillated fibrinous coagulum, enclosing leucocytes in its meshes (Fig. 10). This coagulum, however, is usually not so abundant as it is in the ordinary pneumonic process, neither is the fibrillation quite so distinct. As to how far all the small cells

enclosed within it are emigrants, it is difficult to speak with absolute certainty. Small cells indistinguishable from leucocytes are frequently seen in

Fig. 10.



Acute Phthisis. Showing one of the alveoli filled with fibrinous exudation and leucocytes, and some cellular infiltration of the alveolar wall. × 200.

varying numbers associated with the larger epithelial elements, and many of these are probably the products of epithelial proliferation; but in some cases they are so numerous that I think they must, for the most part, have escaped from the vessels. These small cells are usually slightly granular, owing to the presence of molecular fat.

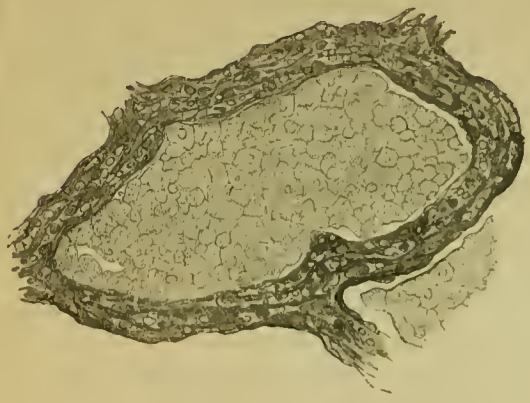
In some of the most acute cases of phthisis, where both lungs are rapidly involved and rapidly

beeome disintegrated, this filling of the air-vesieles with the products of exudation may eonstitute almost the sole cause of the consolidation, the alveolar walls being but little altered. More commonly, however, this change is associated with epithelial proliferation, the two conditions occurring together, just as they have been seen to do in acute miliary tuberculosis. Pulmonary consolidation from exudation is also often met with at the bases of lungs, the upper portions of which are the seats of more ehronic phthisical disease. Here an acute process would seem to have supervened upon a more chronic one, and this often determines the fatal termination. I must, however, state that this acute change in the bases of the lungs can in no way be separated by any marked line of demarcation from less acute proeesses which may be taking place in the upper portions of the organs. It would appear that the element of time constitutes the principal cause of the differences between the two. To this I shall allude again hereafter.

2. *An Accumulation of Large Epithelial Cells within the Pulmonary Alveoli.*—This is one of the most frequent changes met with in phthisis. Large nucleated elements are seen filling the alveolar cavities, and, in successful sections, extending also into the alveolar passages and terminal bronchioles (Fig. 11). These elements resemble epi-

thelium, and are evidently the offspring of the epithelial cells which line the alveolar cavities. They are plump and spheroidal in shape, and although varying somewhat in size, are for the most part about four or five times as large as a white blood-cell. Some of them are much smaller, and appear

FIG. 11.



Acute Phthisis. Showing one of the alveoli filled with epithelial elements, and marked cellular infiltration of the alveolar wall. × 200.

to be less fully developed. They contain a distinct round or oval nucleus with nucleolus, and in some of the larger ones as many as two or even three nuclei are visible. They are usually markedly granular, and sometimes contain dark pigment. Associated with these large cells there are seen a few much smaller elements, many of which are in-

distinguishable from leucocytes. Although some of these may be the offspring of the germinating epithelium, I think they are probably, in part, emigrants from the blood-vessels; emigration being so frequently concerned in the production of phthisical consolidation.

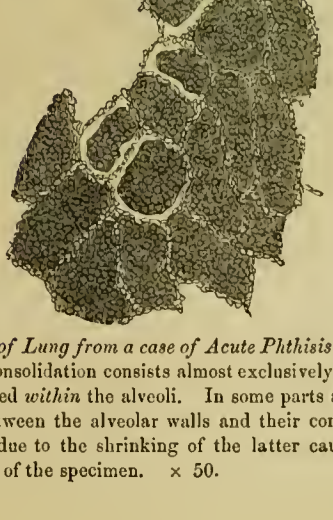
I must here state that it has been questioned by some pathologists whether the accumulation of epithelial cells within the air-vesicles in phthisis is to be regarded as the result of an active (inflammatory) process; and that it has been supposed to be due to a swelling, loosening, and shedding of the alveolar epithelium, such as so constantly occurs in mechanical hyperæmia and œdema of the lung. To this I would reply, that the number of the cells, together with the prominence and occasional multiplicity of this nuclei, appear to me to be tolerably conclusive evidence of the occurrence of new formation. At all events, this epithelial change in phthisis, which is more marked in this than in any other pulmonary lesion, presents a marked contrast to the few scattered detached cells seen in the alveoli in the conditions of pulmonary hyperæmia and œdema alluded to.

In describing these changes within the alveoli, allusion must be made to those large multinucleated masses of protoplasm—the "giant cells"—which are frequently met with in phthisical lungs,

inasmuch as these cells are often seen to be occupying situations which are distinctly intra-alveolar. These cells are precisely similar to those which have been already described as occurring in acute miliary tuberculosis. They are, however, much less commonly found. Respecting their origin and pathological significance, I would refer to what I have already said when treating of that disease. As in tuberculosis so in phthisis, they are not found in the earlier stages of the process.

This accumulation of epithelium within the pul-

Fig. 12.



Section of Lung from a case of Acute Phthisis. Showing that the consolidation consists almost exclusively of products accumulated *within* the alveoli. In some parts a free space is seen between the alveolar walls and their contents: this is simply due to the shrinking of the latter caused by the hardening of the specimen. × 50.

monary alveoli occupies a very prominent position in the histology of phthisis. It is, I believe, almost invariably present in acute cases. In some of those which are the most acute it is often associated with exudation products, as already described; but in others it may constitute the only marked structural change (Fig. 12). Both these forms of intra-alveolar change are, however, invariably associated with more or less cellular infiltration of the alveolar walls; although this infiltration in acute cases is sometimes so slight that it may easily escape observation. In all cases where the alveoli contain confluent epithelial elements, or giant cells, the infiltration of their walls is considerable, and causes marked thickening.

The macroscopical appearances of the lungs in those cases of phthisis in which the pulmonary consolidation is due mainly to these *intra*-alveolar changes, and in which there is but little alteration of the alveolar walls, are very characteristic. The consolidated tissue is quite soft and friable, breaking down very readily under the finger, and there is a complete absence of any induration. The consolidation, although frequently almost uniform, usually presents a somewhat lobulated outline, indicating the implication of different groups of lobules. The colour varies from a reddish to a yellowish-grey, and scattered through the consoli-

dated mass are small portions of a more decidedly yellow tint. These latter correspond with those parts in which the retrogressive changes are the most advanced, and they are even softer in consistence than the surrounding tissue. In many parts the consolidated tissue will be found broken down, so as to form cavities of various sizes. These usually possess irregular walls, which are quite soft and friable, like the solid tissue which surrounds them. Sometimes a cavity is found lined with a soft, delicate membrane.

3. *An Infiltration and Thickening of the Alveolar Walls with small Cells.*—This is undoubtedly the most important and characteristic of all phthisical lesions. It is, I believe, invariably present in all forms of phthisical consolidation of the lung, although its extent varies very considerably in different cases. Its universality has been especially insisted upon by Dr. Wilson Fox, and I entirely agree with this observer as to the prominent position which it occupies in the histology of phthisis.*

The change is precisely similar to that which has been already described as occurring in acute

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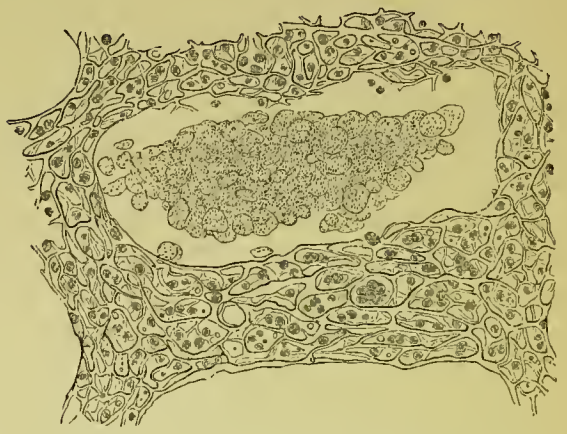
* "Discussion on the Anatomical Relations of Pulmonary Phthisis to Tubercle of the Lung." Dr. Wilson Fox. *Trans. Path. Soc. Lond.*, vol. xxiv. p. 284.

miliary tuberculosis. In its earlier stages, a few small, round, lymphoid cells are seen infiltrating the alveolar septa, which are thus slightly thickened. (See Fig. 11, p. 39). As the change proceeds, the number of these cells increases, and from them an imperfect fibro-nucleated structure is developed which, in

Fig. 13.



Section of Lung from a case of somewhat Chronic Phthisis. Showing the thickening of the alveolar walls by a fibro-nucleated adenoid-like tissue; together with an accumulation of epitbelial cells within the alveolar cavity. The latter are undergoing retrogressive changes. × 200.

some parts, may closely resemble adenoid tissue (Fig. 13). This structure contains no new blood-vessels. As this new tissue develops in the alveolar walls, it gradually obliterates and replaces the alveolar cavities, so that whilst in some por-

tions the thickened alveoli may be found still containing exudation products, epithelial elements, or giant cells, in others large tracts will be seen consisting almost entirely of the small-celled growth. Associated with it there are often varying quantities of black granular pigment.

The development of this new non-vascular growth in the alveolar walls leads to the partial or even complete obliteration of the pulmonary capillaries. The manner in which this obliteration is effected is best observed in vessels situated in the vicinity of the more completely consolidated tissue, where the development of the growth is only commencing to take place. Here the vessels may be seen either partially or wholly surrounded with the small-celled tissue, which, as it increases, appears to compress and gradually to diminish their calibre. This vascular obliteration has been especially insisted upon by Professor Buhl and Dr. Wilson Fox. Allusion will again be made to it subsequently.

The changes which may subsequently take place in this alveolar growth vary. The infiltrated septa may rapidly break down before any marked thickening or development of new tissue has had time to occur; whilst in other less acute cases there is a considerable development of the imperfect fibro-nucleated tissue, which, although it may remain as a more or less permanent structure, usually, owing

to insufficient vascular supply, undergoes in its turn retrograde metamorphosis. These two kinds of change are very often found taking place simultaneously in different portions of the consolidated lung. In those portions in which the new tissue is undergoing degeneration, it, together with the cells which may be contained within the alveoli, will be seen to have become converted into a structureless granular débris in which the outline of the cellular elements is no longer distinguishable, whilst perhaps in immediate vicinity to these more degenerated portions will be found a more permanent fibro-nucleated structure.

Respecting the alteration which this alveolar growth produces in the physical characters of the pulmonary consolidation, it may be stated generally that it usually leads to more or less induration. The extent of this induration, however, will vary according to the characters of the new tissue. If the tissue be almost entirely cellular, such as it is in its earlier stages and when it is very rapidly developed, it will produce but little, if any, induration of the pulmonary consolidation, which, consisting mainly of the intra-alveolar accumulations, will be soft and friable in consistence, much resembling that which has been already described. When, on the other hand, as is more frequently the case, there is any considerable development of the im-

perfect fibro-nucleated growth, there will be a corresponding induration of the consolidated tissue. In many cases these changes produce uniform tracts of indurated consolidation of a greyish colour mottled with black pigment, in which there may be scattered here and there yellowish patches corresponding to those portions which have undergone retrogressive fatty changes.

4. *An Increase in the Interlobular Connective Tissue.*—This is met with to a greater or less extent in all the more chronic forms of phthisis. By the interlobular connective tissue I mean that tissue more particularly which supports the bronchi and larger blood-vessels. It is obvious that between this and the more delicate structure which constitutes the walls of the air-vesicles there is no histological line of demarcation; the one passes imperceptibly into the other. At the same time it is advisable to speak of them separately in describing morbid changes in the lung, inasmuch as not only does the part which they respectively play in the production of the pulmonary consolidation differ in different cases of phthisis, but the new growths to which they give origin present certain differences both in their histological characters and in their pathological tendencies.

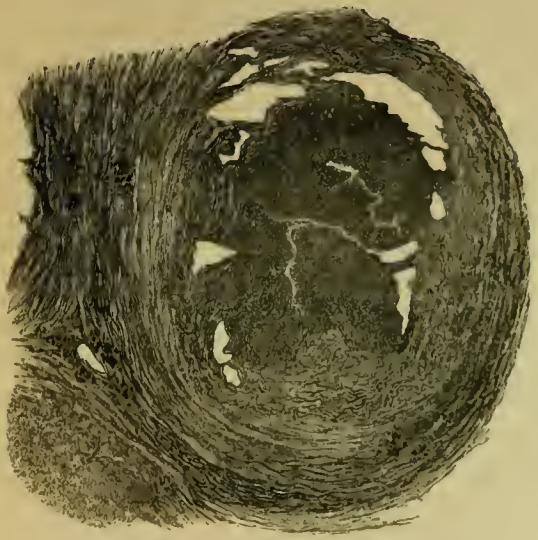
With regard to the histological characters of the new growth which originates from the interlobular

tissue—although in the earlier stages of its development, when it is richly cellular, many parts of it may resemble the growth in the alveolar walls—its structure, for the most part, is like that which is met with as the result of chronic indurative processes in other organs. It has a much greater tendency to become developed into a fibroid tissue than the alveolar growth, and is much less frequently the seat of secondary retrograde changes. As usually met with, it consists either of wavy fibres or of a more or less reticulated structure, with a varying proportion of round, spindle-shaped, or branched cells (Fig. 14). Associated with it, in most cases, are granules of black pigment. These differences in the pathological tendencies and structure of the alveolar and interlobular growths are, probably, mainly owing to differences in the amount of their vascular supply. Whereas in the former the vessels become obliterated in the manner already described, in the latter this obliteration is much less complete or entirely wanting.

An increase in the interlobular tissue in phthisis, inasmuch as the new tissue has so marked a tendency to become fibroid, leads to extensive induration of the pulmonary texture; and further, owing to the contraction which the tissue tends to undergo, its growth ultimately produces a corresponding contraction of the diseased lung. In all cases of phthisis,

in which there is either a marked thickening of the alveolar walls or an increase in the interlobular connective tissue, any cavities which may exist in

Fig. 14.



Chronic Phthisis. Showing the new interlobular fibroid growth surrounding and encapsulating a degenerated and caseous portion of the consolidated lung. × 50, reduced ½.

the consolidated and indurated lung are characterised by the tough and fibroid character of their walls, these presenting a marked contrast to the soft friable tissue which surrounds the cavities in those cases in which the pulmonary consolidation is mainly due to intra-alveolar changes.

Changes in the Bronchi.—I must now allude to certain changes in the bronchi, as these tubes are invariably more or less involved in pulmonary phthisis. The changes which takes place in them are mainly of two kinds—a superficial catarrhal process, and an inflammatory process implicating the deeper structures of the mucous membrane and the peri-bronchial tissue.

More or less catarrh of the bronchi is constantly present in phthisical lungs. Sometimes this catarrh is general, affecting the tubes throughout the whole of both lungs, although the phthisical consolidation may involve only a much smaller area. This is especially the case in some acute forms of phthisis. In such lungs the bronchi often exhibit universally well-marked signs of an acute catarrhal process, and the smaller tubes may be filled with a tenacious puriform secretion. Much more commonly, however, the catarrh is limited, and more strictly confined to such portions of the lungs as are becoming, or have already become, consolidated.

The appearances presented by the bronchi, and the characters of the secretion which they contain, will obviously differ according to the severity and duration of the catarrhal process. In the most acute cases, in which the catarrh has been only of short duration, beyond more or less increased vascularity of the mucous membrane and the presence of an abundant secretion rich in cellular elements,

there is little that is abnormal discoverable. In those cases, on the other hand—and these are the most frequent—which are of longer duration, and in which the changes in the bronchi are more particularly confined to the consolidated portions of the lung, the alterations are more marked; and it is in such cases that the deeper structures of the bronchial wall so constantly become involved.

In speaking of this implication of the deeper structures of the bronchial wall, I must ask you to bear in mind what I have already stated respecting that marked tendency to a cellular infiltration of the sub-epithelial connective tissue which exists in inflammations of mucous membranes occurring in scrofulous persons. Although such infiltration certainly occurs also to a less extent in all inflammations of mucous membranes which become chronic, it supervenes earlier, is much more marked, and is more destructive in the scrofulous; and, inasmuch as it is in such that phthisis is especially common, it occupies a prominent place amongst the changes we are describing.

If the smaller bronchi be examined microscopically, numerous round cellular elements, many of which are of relatively large size, will usually be found extending from the epithelium into the sub-epithelial connective tissue. (See Fig. 9, p. 27.) As the result of this infiltration the membrane looks

swollen and somewhat opaque. The epithelium often becomes loosened and detached, thus giving rise to superficial irregular-shaped erosions; and the sub-epithelial infiltration may disintegrate, and so lead to the production of small ulcers. The latter is especially the case in those who are markedly scrofulous. Ulceration, however, in my experience, is less common than has been represented by some other observers. Professor Rindfleisch describes it as of very constant occurrence.* I have examined the bronchi in a large number of cases, and I should be inclined to say that ulceration is the exception rather than the rule. The ulcers have thickened opaque edges, and when once formed they usually extend, so that ultimately they may involve considerable areas of the bronchial wall.

In addition to this cellular infiltration of the bronchial wall, there is very frequently a similar infiltration of the peri-bronchial tissue. In this situation some small nodules of cell growth are often found surrounding the smallest bronchi. From the description of similar nodules which are stated by Dr. Burdon Sanderson and Dr. Klein to exist largely in the lungs of tuberculous animals, I presume that they are produced by a proliferation of the lymph-

* Ziemssen's "Cyclopædia of the Practice of Medicine," vol. v. p. 661.

follieles which are situated in the walls of the peri-bronehial lympathies. These lymph-follicles are described by Dr. Klein as existing in the wall of the lymphatic vessel between the bronchus and the accompanying branch of the pulmonary artery.* It seems probable, as stated by Rindfleisch, that the development of these nodules is due to the transmission of infeective substanecs by means of the lymphaties from thc bronehial walls. The peri-bronchial growth, although it usually disintegrates, may undergo more or less imperfect fibroid develop-ment. In the former case, this disintegration going on hand in hand with that of the adjacent pulmonary consolidation, a eommunieation may bc established between the two through the bronehial wall.

Having deseribed thc morbid proeesses which take plaee in the bronchi, it remains to consider the alterations whieh these processes may produee in the adjacent pulmonary tissue. These alterations are important. In the prcsent plaee, however, I propose merely to allude to such histological ehanges in the lung as may result from the infiltration of the bronchial walls. The interference with the inflation of the air-vesicles, and the im-

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* "The Anatomy of the Lymphatic System." By Dr. E. Klein. Part II. Chap. iv.

pediment to the circulation through the pulmonary capillaries, which may be induced by any concomitant bronchial obstruction, will be considered subsequently.

In order to understand the alterations which the cellular infiltration of the smaller bronchi may produce in the pulmonary tissue, it is important to bear in mind the direct histological continuity which subsists between the peri-bronchial and interlobular connective tissue, and also between the latter and the air-vesicles. The infiltration of the bronchial and peri-bronchial tissue, which has been seen to be so frequent in phthisical lungs, gradually invades the interlobular tissue with which it is directly continuous, and the new interlobular growth in its turn invades the alveoli, producing a thickening of their walls, and ultimately completely obliterating them. There is thus a gradual invasion of the lung by a small-celled growth having its origin in the walls of the smaller bronchi; and the new growth not only extends laterally, but it also invades the air-vesicles at the termination of the bronchial tube.

Whilst the development of a small-celled growth having its origin in the bronchial walls may thus contribute to the consolidation of the lung, as it does in some forms of pulmonary induration which do not come within the category of phthisis (*e.g.*,

the consolidation so often resulting from long-continued bronchitis, Corrigan's cirrhosis, &c.), it must be borne in mind that in most cases of phthisis this consolidation is principally due to those processes which have already been described as commencing in the pulmonary alveoli. How far the bronchial growth may be a cause of the alveolar changes will be considered in a subsequent lecture.

THE PATHOLOGY OF PHTHISIS.

Thus far the consideration of phthisis has been strictly limited to a description of the several structural changes which are met with in the lungs. I propose now to consider, in the first place, the nature of the morbid processes upon which these changes depend, and subsequently to discuss the etiology of the disease.

The nature of the morbid processes which give rise to that disintegrative consolidation of the lungs which constitutes phthisis has long been one of the most vexed questions in pulmonary pathology, and yet the answer to this question must have a most important bearing upon the prevention and successful treatment of this disease. It would be beyond the scope of the present lectures to attempt to discuss the numerous doctrines which have been held from time to time by different

pathologists respecting the nature of the pulmonary lesions in phthisis. These doctrines, however, as I have already indicated, have obviously varied according to the different meanings which have been attached to the term "tubercle." According to the older views, which were based upon the teachings of Bayle and Laennec, tubercle was regarded as a specific non-inflammatory growth which was characterised by the caseous degeneration which it invariably underwent; and this caseous metamorphosis was held to be such a distinguishing peculiarity of the growth that all caseous masses came to be regarded as tubercular; and phthisis, in which caseation plays such a prominent part, was consequently regarded as a tubercular disease. Somewhat later, Lebert and others endeavoured to support this then current doctrine by microscopical investigation. Lebert stated that tubercle possessed a definite structure—that it consisted of what he termed "tubercle-corpuscles;" and these corpuscles were thought to be in themselves quite characteristic.

This doctrine of the specificity of tubercle and phthisis was almost universally accepted up to the time of Virchow, by whose teaching it became very materially modified. Virchow showed that caseous matter was by no means necessarily tuberculous, but that it might originate from the fatty degene-

ration and inspissation of many morbid products. He consequently framed a new definition for "tubercle," and limited the application of the term to the grey granulation, which he still regarded as a non-inflammatory growth. With this limitation it became obvious that very much of the pulmonary consolidation met with in phthisis was not due to tubercle, and that although grey granulations were frequently found in phthisical lungs, the production of the large tracts of consolidation which had formally been described as "infiltrated tubercle" must be accounted for in some other way. These tracts of consolidation, and, in short, nearly all the varieties of phthisical consolidation which did not assume the form of the grey granulation, were regarded by Virchow as inflammatory in their origin, and were described by him as scrofulous or caseous pneumonias.

This view of the origin of phthisical consolidation was subsequently advocated with great ability by the late Professor Niemeyer. Niemeyer not only regarded the consolidation as inflammatory in its origin—as the result of what he termed a catarrhal pneumonia—but, accepting the doctrine of the infective nature of tubercle which had been promulgated by Buhl, he stated that tubercle was merely a secondary and *accidental* accompaniment of the disease.

The older doctrine of the tubercular nature of phthisis, on the other hand, has received strong support in our own country from Dr. Wilson Fox, and more recently abroad by Professor Rindfleisch. Dr. Fox states, as the result of his prolonged and well-known researches in pulmonary pathology, that the disintegrative consolidation of the lungs in phthisis is almost invariably associated with the development of a small-celled growth in the alveolar walls, which he regards as tubercular.* This so-called tubercular growth he considers to occupy a different pathological position from any epithelial proliferation or exudation which may have taken place within the alveoli. The latter he regards as inflammatory products, whilst the tubercular growth he believes to be the principal cause of that obstruction to the capillary circulation, and consequent disintegration of the pulmonary consolidation, which is so characteristic of the disease.

There has been a very general tendency during recent years to subdivide phthisis into several different varieties—to attempt to show that it may be produced by different morbid processes. A "pneumonic" phthisis, a "tubercular" phthisis, a "scrofulous" phthisis, and a "fibroid" phthisis, have been described as representing distinct patho-

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* *Loc. cit.*

logical varieties of the disease. Great diversity of opinion respecting the nature of phthisis thus exists amongst pathologists even in our own time; and whilst some regard tubercle as the most important element in all cases of the disease, and others maintain that it is only an accidental accompaniment, a third class of observers tell us that some cases of phthisis are tubercular, but that others are not. These diverse doctrines, as I have already stated, are in great measure to be accounted for by the want of agreement existing amongst pathologists as to what constitutes pulmonary tubercle. This question I have attempted to answer. I proceed now to consider the nature of the morbid processes met with in the lungs in phthisis, and to point out how far these processes resemble those which give rise to the disseminated lesions of acute miliary tuberculosis.

In order to determine the nature of the morbid processes which give rise to the various histological changes met with in the lungs in phthisis, it will be necessary to compare these changes, in the first place, with those occurring in morbid conditions of the lung which do not come within the category of this disease, and the pathology of which is comparatively well understood; and, secondly, with any apparently similar changes which may be met with in other organs.

If the histological changes of phthisis be considered separately, it will be found that they are, for the most part, similar to those occurring in other well-known pulmonary diseases. The most important changes met with in phthisical lungs have been seen to be of four kinds:—1st. The presence of leucocytes and exudation matter within the alveoli; 2nd. An accumulation within the alveoli of large cellular elements, which are the offspring of the alveolar epithelium; 3rd. A cellular infiltration and thickening of the alveolar walls; and 4th. An increase in the interlobular connective tissue. These four kinds of change, which may be associated in very different degrees, constitute the most important factors in the production of the phthisical consolidation.

Firstly, with regard to the presence of leucocytes and exudation matter within the pulmonary alveoli:—That this is quite frequent I have already insisted upon, and in some of the most acute forms of phthisis it may constitute the prominent structural change. Between these exudation products and those which are met with in ordinary croupous pneumonia there is, in a large number of cases, no histological distinction; and although, in other cases, the leucocytes may be somewhat less and the epithelial products somewhat more numerous than is usual in simple pneumonia, the two changes so

closely resemble one another that they must clearly both be regarded as the results of an acute inflammatory process. Why the two processes should differ so widely in their ultimate results—the one usually terminating in complete resolution, whilst the other leads to the disintegration of the lung—I will attempt to explain subsequently. At present I am merely desirous to establish the position that, in certain cases of phthisis, the disintegrative consolidation of the lung is the result of an acute pneumonic process—a process which produces histological changes similar to those met with in ordinary croupous pneumonia.

A more important cause of phthisical consolidation of the lung than simply the presence of exudation products is the accumulation within the alveoli of large cellular elements derived from the alveolar epithelium. These two kinds of change, as already stated, are frequently associated, although the epithelial growth is certainly in the majority of cases the predominant lesion, and in many forms of acute phthisis it may be almost the sole cause of the pulmonary consolidation. A similar epithelial proliferation occurs in other morbid conditions of the lungs, although rarely to such an extent as it is met with in phthisis. In what is generally known as broncho- or catarrhal-pneumonia, there is often a proliferation and accumulation of epithelium

within the pulmonary alveoli. In making this statement, however, I must add that many of the disseminated patches of consolidation which are included under this head are merely partially collapsed groups of air-vesicles which have become filled with mucus inhaled from the bronchi, and that, although the alveolar epithelium may have become loosened, it exhibits no signs of active growth. A certain amount of epithelial proliferation frequently occurs also in ordinary croupous pneumonia, and especially in the secondary pneumonias which are often met with as the acute process terminating a chronic disease. In these secondary pneumonias, in which the inflammatory process is usually of less intensity than in the primary forms, the epithelial elements are sometimes so abundant that they apparently play as prominent a part in the production of the consolidation as the leucocytes and exudation matter with which they are associated. Lastly, in many cases of acute miliary tuberculosis, the pulmonary lesions are characterised by epithelial activity. It is thus evident that active changes in the alveolar epithelium constitute a part of the tissue-changes met with in pulmonary inflammation; and we are, I think, justified in concluding that similar changes occurring in phthisis, associated as they so frequently are with exudation, are also the result of an inflammatory process.

In considering the cellular infiltration of the alveolar walls, it is in the first place to be remembered that it is precisely similar to that which occurs in acute tuberculosis. In non-phthisical and non-tubercular forms of pulmonary disease this is not nearly so frequently met with as are the other changes occurring in phthisis, and it must certainly be regarded as the most distinctive histological change. Its relation to scrofula I have already alluded to, and on this head I shall have more to say hereafter.

There are, however, certain inflammatory conditions of the lung which do not come within the category either of acute tuberculosis or of phthisis, in which a similar kind of change occurs in the alveolar walls. In certain cases of ordinary basic croupous pneumonia, in which the exudation products are not readily absorbed and the condition becomes more or less chronic, the walls of the alveoli will be found thickened by a cellular growth. In such cases the lung is less friable, tougher, and more pigmented than in ordinary hepatisation. Then, again, in those conditions of the lung which more properly come within the category of what is usually known as chronic pneumonia, and of which Corrigan's cirrhosis may be taken as the type, there is invariably a development of small-celled tissue in the walls of the air-vesicles. Lastly, I must mention that a similar growth is developed around the

smaller bronchi, from which it gradually extends to, and invades the walls of, the alveoli, in many cases of long-continued bronchial catarrh. The new growth in all these cases is often quite similar to that met with in phthisis, although it presents, for the most part, a greater tendency to become organised and developed into an imperfect fibre tissue.

Respecting the remaining structural change met with in phthisis—an increase in the interlobular connective tissue—it is only necessary to remark that a similar increase occurs, to a greater or less extent, in all conditions of long-continued pulmonary irritation. In the disease known as Corrigan's cirrhosis this kind of change reaches its maximum. When occurring in phthisis it is also to be regarded as the result of a chronic inflammatory process, and its extent bears, for the most part, a direct relation to the chronicity of the disease.

I have thus endeavoured to show, in the first place, that the several structural alterations met with in the lungs in phthisis are the results of morbid processes which must be regarded as being inflammatory in their nature; and, secondly, that these alterations are by no means peculiar to phthisis, but are precisely similar to those met with in other forms of pulmonary inflammation. In

describing the morbid processes as inflammatory, I merely mean to imply that they owe their origin to some kind of injurious irritation. It has already been stated that the several kinds of change are usually associated, although in such different degrees that, whilst in some cases of phthisis the pulmonary consolidation is due almost entirely to one kind of change, in others the predominant lesion is entirely different. Admitting, therefore, the inflammatory nature of the lesions, it remains to consider the causes of these differences in the mode of their association.

In order to explain the differences in the kind of lesion which predominates in different cases of phthisis, it will be necessary to bear in mind certain points connected with the pathology of the various textural alterations which accompany the process of inflammation. It must be remembered that the injury which is the starting-point of every inflammatory process appears to exercise its influence mainly upon the blood-vessels, and that upon alterations which are thus produced in the vascular walls the stagnation of the blood-stream, together with the emigration of leucocytes and exudation of liquor sanguinis, depend. Further, that the greater the severity of the initial injury the greater will be the damage inflicted upon the walls of the blood-vessels, and consequently, *cæteris*

paribus, the more abundant the exudation and emigration. There are, however, in addition to these vascular phenomena, certain changes in the tissue outside the vessels, and it is respecting the nature of these extra-vascular changes that I would now more especially speak.

In studying the alterations which take place in inflamed tissues, it will, I think, be seen that they vary considerably according to the intensity and duration of the inflammatory process. The relation which appears to me to subsist between the intensity and duration of the inflammation and the resulting textural changes, I would express in terms similar to those which I have employed elsewhere—viz., the less intense and more prolonged the inflammatory process the more do the resulting textural changes tend to be limited to the connective tissue which is immediately adjacent to the blood-vessels and lymphatics; whereas, in inflammations of somewhat greater intensity more distant elements become involved. Further, the new elements in the tissue immediately adjacent to the blood-vessels and lymphatics have a special tendency to become developed into a fibroid structure; hence the induration of the organ, which is such a constant result of chronic inflammation. The truth of these propositions is more particularly manifest in those structures which possess epithelium—as,

for example, in the kidneys, mucous membranes, and in the lungs. In the kidneys, the more severe forms of inflammation (excluding those which are the most severe, and which are attended by the formation of abscess) are characterised anatomically by swelling and (probably) proliferation of the tubal epithelium; and it is only when such inflammations become less severe, and pass into a chronic form, that the peri-vascular connective tissue becomes extensively involved; whilst in the least severe and most chronic forms of inflammation, increase of this tissue is almost the only active structural change. In mucous membranes, also, the more severe inflammations are attended by epithelial proliferation; the less intense and more chronic, by an increase in the sub-epithelial connective tissue.

In the lungs, with which we are now more especially concerned, this relation between the intensity of the inflammation and the resulting textural changes is even still more apparent. Excluding those most intense and concentrated forms o pulmonary inflammation which lead to the formation of abscess, the most severe form of inflammation met with in the lung is what is commonly known as croupous pneumonia. In this form of pneumonia, exudation and emigration play the most prominent part, although there may be more

or less increased activity of the alveolar epithelium. Of pulmonary inflammations, those which come next in order of intensity are the catarrhal pneumonias, and those constituting many of the more acute forms of phthisis. In some of those cases of phthisis which are the most acute the pulmonary consolidation, as already stated, may consist almost entirely of exudation products; but more commonly epithelial proliferation constitutes the predominant structural change.

If we pass on to consider those forms of pulmonary inflammation which are characterised by more or less chronicity—whether the pneumonic process was of slight intensity from its commencement, or is the sequence of a more acute attack—it will be found that they are invariably attended by some change in the walls of the alveoli. This change consists in a cellular infiltration and thickening of the alveolar wall, in many respects similar to what has been already described. Such thickening occurs in those exceptional cases of basic croupous pneumonia which pass into a chronic stage, and in those other non-phthisical forms of pulmonary consolidation which are commonly described as chronic pneumonias. It is, however, in phthisis that this alveolar change is so constantly met with, and although the character of the new growth here, as will be seen presently, usually differs from that of

non-phthisical inflammation, its extent bears, for the most part, a direct relation to the chronicity of the disease.

Another result of chronicity in pulmonary inflammation is an increase of the interlobular connective tissue. Active changes in this tissue usually occur later in the course of chronic inflammation than do the changes in the alveolar walls, and they must be regarded as evidence of greater chronicity in the inflammatory process. In the most chronic cases of pneumonia, phthisical and non-phthisical, this tissue becomes considerably increased; and this increase is so marked in many of the most chronic forms of phthisis that these have been classified as a distinct variety of the disease under the name of "fibroid phthisis."

If these points in the pathology of inflammation be applied to phthisis, they will, I think, help to explain the differences met with in the kind of lesion which predominates in different cases of this disease. Here also the intensity of the inflammatory process determines to a great extent the nature of the resulting textural changes; and in using the term "intensity," I would again state that by it I wish to be understood to imply two factors—severity of injury, and susceptibility of tissue injured. In those forms of phthisis in which the process is of maximum intensity, the consolidation of the lung

being the most rapidly induced, exudation and emigration may occupy a prominent place, although, as already stated, such cases are comparatively rarely met with. In cases of somewhat less intensity, epithelial proliferation will take a larger share in the production of the consolidation, and there will be more marked changes in the alveolar walls; whilst in those cases in which the inflammatory process is least intense and most chronic, the growth in the alveolar walls and interlobular tissue will constitute the predominant lesions.

LECTURE III.

THE PATHOLOGY OF PHTHISIS, ETC.

Secondary changes in the pulmonary consolidation—Resolution—Development into an imperfect fibroid tissue—Retrograde metamorphosis due to four causes—Secondary inflammation and ulceration of the lung—Etiology of Phthisis—Inherent pulmonary weakness inherited or acquired—Its influence on pulmonary inflammations—Methods by which injuries may be inflicted on the lungs—Chilling of surface of the body—Injuries inflicted through the medium of the bronchi—Bronchial catarrh as a cause of Phthisis—Bronchial inflammation may lead to pulmonary consolidation in three ways—Direct injury of air-vesicles—Nature of injury—Blood in bronchi as a cause of Phthisis—Lobulated distribution of phthisical consolidation—Infection—General health of the individual—Why are the apices of the lungs the first to become involved?—Pathological varieties of Phthisis—Syphilis and Phthisis—Influence of pathology upon the treatment of Phthisis.

GENTLEMEN,—Thus far our consideration of the pathology of the pulmonary lesions met with in phthisis has been confined to an attempt to determine their nature, and to account for the differences in their mode of association. It remains to consider the secondary changes which the several kinds of lesion may respectively undergo. It may be stated generally

that the secondary changes which occur in the pulmonary consolidation of phthisis are of three kinds—*resolution, development into an imperfect fibroid tissue*, and *retrograde metamorphosis*. Some of that consolidation of the lung which is the most rapidly induced, and which is consequently mainly due to the presence of intra-alveolar exudation matter, may become absorbed. The resolution of the consolidation may thus be complete, or after the absorption of the intra-alveolar products there may remain more or less infiltration of the alveolar walls. The two last-named kinds of change produce that induration on the one hand, and caseation and softening on the other, which associated in such various degrees make up the diverse physical characters of the phthisical lung.

Firstly, with regard to the development into a fibroid tissue. This, as has already been seen, may take place from the small-celled growth in the alveolar walls and from the interlobular structures. The new tissue which originates in the walls of the alveoli, being for the most part destitute of blood-vessels, is incapable of forming a mature structure. In those cases of phthisis, however, in which the inflammatory process is of slight intensity, much of the cellular infiltration of the alveolar septa becomes developed into an imperfect fibroid or adenoid tissue, which may remain for some time permanent, and

so contribute largely to the induration of the lung. But the new growth originating in the interlobular tissue is the principal source of the pulmonary fibrosis. In this there is not the same vascular obliteration, and hence it forms a much more fully developed and permanent structure. The amount of fibrosis, I would again repeat, is *cæteris paribus* in direct proportion to the chronicity of the disease.

The other kind of secondary change met with in phthisis is the retrograde metamorphosis of the consolidated lung. It is this kind of change which leads to that caseation, softening, and disintegration which is so characteristic of the disease, and which distinguishes phthisical from other forms of pneumonic consolidation.

A retrograde change in the inflammatory products is an invariable accompaniment of acute non-phthisical pneumonia. Much of the exudation matter and epithelium which fills the air-vesicles undergoes fatty degeneration, and as the circulation becomes restored in the pulmonary capillaries the fatty débris is removed by absorption, and the lung remains intact. In phthisical consolidation, however, this rapid removal of the inflammatory products does not take place. The contents of the alveoli undergo fatty degeneration, but the degenerated products are not removed, the infiltrated alveolar walls are destroyed, and the consolidated

lung undergoes a rapid or gradual process of disintegration.

In studying the causes of this retrograde change, which is so distinctive of phthisis, I think it is evident that they are of four kinds :—

1. Certain histological conditions of the consolidated tissue which tend to interfere with its vascular supply.

2. The absence of any formation of new blood-vessels in the small-celled growth which originates in the alveolar and bronchial walls.

3. Secondary inflammation and ulceration of the lung.

4. An inherent weakness of the lungs (usually inherited), which not only renders them especially susceptible to injury, but, when injured, renders them abnormally incapable of recovering themselves from the inflammatory process which has been induced.

I will speak firstly of those histological conditions which tend to interfere with the circulation. It is this interference with the circulation which undoubtedly constitutes the most important element in the causation of the pulmonary disintegration. Of these conditions, that which occupies the most prominent place is that cellular infiltration of the walls of the alveoli and smaller bronchi which we have seen to be such a constant, though very variable, factor in phthisis. I have already alluded

to this infiltration as a characteristic of scrofulous inflammation, and have pointed out that it occurs in a modified form in those who are not markedly scrofulous, and also in all pulmonary inflammations which become chronic. When the infiltration is marked, and especially when rapidly induced, the effect of the pressure which the young cells exercise upon the pulmonary capillaries is to obstruct the circulation, and so not only to prevent the absorption of any intra-alveolar products, but also to lead to necrotic changes. This infiltration—the most distinctive histological feature of phthisis—is the most important cause of the disintegration of the lung.

There are two other conditions which, although of much less importance than the preceding, also tend to interfere with the circulation and so to cause necrosis. These are the pressure which is exercised upon the pulmonary capillaries by the inflammatory products which have accumulated within the alveoli, and that tendency to stagnation of the blood-stream which is an invariable accompaniment of every acute inflammation. The operation of intra-alveolar pressure obtains in those cases of phthisis which are the most acute. Here the exudation matter and epithelial elements often accumulate so as to fill and even distend the alveoli, and in so doing they must necessarily com-

press more or less the capillary blood-vessels. That tendency to stagnation of the blood-stream which exists in all severe inflammations must also constitute an element in the causation of the necrosis in these acute forms of the disease.

Respecting the second cause of the pulmonary disintegration which I named—the absence of any new formation of blood-vessels in the small-celled growth which originates in the alveolar and bronchial walls—it is only necessary to remark that this absence of vascular formation must account to a great extent for the retrograde metamorphosis of the lung in those less acute forms of phthisis in which the development of this small-celled tissue contributes so largely to the pulmonary consolidation.

By secondary inflammation and ulceration of the lung, I mean inflammation resulting from the irritation of retained secretions and inflammatory products, which leads to an ulcerative destruction of the bronchial and alveolar walls, and of the surrounding indurated tissue. This occurs in chronic pneumonia with bronchiectasis, and it constitutes an element in the causation of disintegration in some of the more chronic forms of phthisis, but especially in those cases in which there is no marked constitutional state, and in which the disease is exceedingly localised. In such cases the cellular infiltra-

tion of the alveolar and bronchial walls often leads to the formation of an imperfect fibrous or adenoid structure, and much of the disintegration is due not to primary necrosis, but to a gradual ulcerative process which extends centripetally from the terminal bronchi.

Of inherent weakness of the lung as a cause of pulmonary disintegration, I shall speak when considering the etiology of the disease.

THE ETIOLOGY OF PHTHISIS.

Thus far in our consideration of phthisis we have been led to conclude that the various kinds of pulmonary consolidation are the results of inflammatory processes, and that the differences in the variety of lesion which predominates, together with the kind of secondary change which takes place in the consolidated tissue, are mainly owing to differences in the intensity and duration of the inflammation. In describing the several lesions as *inflammatory*, I would again repeat that I merely mean to imply that they owe their origin to some kind of injurious irritation of the pulmonary tissues. We proceed now to consider more particularly the etiology of the disease.

In studying the etiology of pulmonary phthisis it will be necessary to consider, in the first place,

that inherent weakness of the lungs which exists so frequently in this disease, and to see how far this weakness explains the marked susceptibility of the organs to injury, and those peculiarities which have been seen to exist in the histological characters of the textural alterations and in the subsequent changes which take place in the consolidated tissue; secondly, to examine into the several methods by which injuries may be inflicted on the lung in such a way as to set up an inflammatory process; and, lastly, to point out in what way the occurrence and progress of the disease are influenced by the general health of the individual.

Firstly, with regard to the inherent weakness of the lungs. That some abnormal condition of the lungs which is quite independent of any material change, or at all events of anything either chemical or anatomical which we are able to recognise, is present in a large proportion of the cases of phthisis, will, I presume, be very generally admitted. This weakness is in most cases an inherited one, and it is usually spoken of as a "constitutional tendency" or "predisposition."

Inherent pulmonary weakness is most frequently a part of that general constitutional state known as scrofula, in which the mucous membranes generally, and especially that of the respiratory organs, are so abnormally liable to become inflamed. In

many cases of phthisis the scrofulous constitution is marked, and the phthisis is associated with other characteristic scrofulous lesions. In others, the scrofulous tendency is much less pronounced, and the lungs alone may suffer. Although so often associated with scrofula, it must, I think, be also allowed that a similar inherent weakness, leading to similar results, not infrequently exists in individuals who would not generally be regarded as scrofulous, who are not prone to scrofulous inflammations, and in whom the lungs alone appear to be at fault.

Although pulmonary weakness is usually inherited, it may undoubtedly also be acquired. It appears to me to be probable that it may be induced by previous attacks of pulmonary inflammation—that a lung which is constitutionally strong may thus be rendered weak, although the inflammatory process has left behind it no recognisable structural change. It must also be borne in mind, that a weakness of the lungs may exist in common with a weakness of the tissues generally. Anything which lowers the standard of health in the individual—such as insufficient food, bad air, previous disease, &c.—produces a general weakness in which the lungs participate, quite irrespectively of any *special* predisposition of these organs; and should any special predisposition exist, such impairment of

general health would greatly favour its manifestation. This will be again alluded to when speaking of the general health as influencing the occurrence and progress of phthisis.

The most important result of this inherent weakness of the lungs is that it renders them abnormally susceptible to the various kinds of injurious irritation, and they are consequently especially liable to become the seats of inflammatory processes. The frequency with which people who inherit this weakness of the lungs suffer from attacks of bronchial or pulmonary inflammation of varying degrees of intensity is a matter of everyday clinical experience. Such people are usually spoken of as "delicate;" they are very susceptible to cold, it is their respiratory organs that suffer, and ultimately they often become phthisical.

Another consequence of this pulmonary weakness, almost equal in importance to the preceding, and one to which I have already alluded, is that the lungs are less capable of recovering themselves from the effects of an inflammatory process than are healthy organs. The inflammation has a greater tendency to become chronic. If active changes cease they are very readily reinduced, the new tissue has less power to undergo progressive (fibroid) development, and the tendency to retrograde change is more marked.

This tendency to retrograde change, the essential feature of phthisical consolidation, we have already seen to be due to various causes. Of these the most important is that cellular infiltration of the alveolar and bronchial walls which is so characteristic of scrofulous inflammations, and which occurs in its most marked form in those cases of phthisis in which there exists that inherent pulmonary weakness which is associated with this constitutional state. An infiltration similar in kind, although less in degree, we have seen to occur also in all inflammations of the bronchial or pulmonary tissues which are *protracted;* and inasmuch as any inherent weakness of the lungs renders these organs not only abnormally prone to become inflamed, but abnormally incapable of restitution, it is obvious that such weakness must constitute an important element in the causation of the retrograde change in those cases also in which scrofula is absent.

I proceed now to consider the several methods by which injuries may be inflicted on the lungs in such a way as to set up an inflammatory process (excluding wounds and surgical injuries). These methods are three in number:—1st. By injuries inflicted upon the surface of the body; 2nd. By injuries inflicted through the medium of the bronchi; and 3rd. By infection.

By injuries inflicted upon the surface of the

body, I mean that general chilling of the surface which results from exposure to cold. That such chilling frequently causes inflammation of some internal organ is well known. Whether it does so by injuring the superficial sensory nerves, and the impression thus received is reflected from these to the internal organ; or whether by lowering the temperature of the blood and producing internal congestion; or by checking the exhalation from the skin, and so causing an accumulation in the blood of products which should be eliminated, and these products act as direct irritants to the organ—it is not my purpose now to discuss. I am merely desirous to state that such superficial injury is one of the means by which inflammatory processes may be induced in the lungs in common with other internal parts. Which organ suffers as the result of the injury appears in great measure to depend upon predisposition or *weakness*. In some people one organ is especially liable to become inflamed; in others, another. If the lungs be the weak organs, as they so very frequently are in phthisis, the exposure to cold may be followed by a bronchial or alveolar catarrh, a pulmonary congestion, or a more intense pneumonic process; and the inflammatory affection may lead to pulmonary disorganisation.

A much more important cause of inflammatory

processes in the lungs than the preceding are injuries inflicted through the medium of the bronchi. The bronchi, communicating as they do with the external air, are especially liable to injury, and, as a result of such injury, a bronchial or alveolar inflammation may be very readily induced. The question which here concerns us is how far inflammation of the bronchial mucous membrane alone may be a cause of phthisis. That such inflammation, whether induced by chilling of the surface of the body or by a more direct bronchial injury, is a frequent precursor, accompaniment of, and even determining cause of phthisical consolidation of the lung, is well known, although opinions differ as to its etiological importance. Some observers state that catarrh of the bronchial mucous membrane can under no circumstances be a cause of phthisical disease, whilst others maintain that it is a most important one. For my own part, I doubt whether bronchial catarrh causes phthisis, except under exceedingly exceptional circumstances, in the absence of a *predisposition* to the disease. Given the predisposition, however, such catarrh becomes a most important causative element.

In considering bronchial catarrh as a cause of phthisis, it is important to bear in mind how greatly the occurrence and character of this catarrh are influenced by the existence of scrofula. Scrofula, as we

have seen, predisposes to catarrh, renders it obstinate, and leads to a deep infiltration of the mucous membrane. The presence or absence of scrofula must consequently materially modify the liability to any changes which may be produced in the lung as the result of bronchial inflammation.

Bronchial inflammation, as is well known, may lead to consolidation of the lung. It may do so in three ways, two of which are exemplified by what occurs in the ordinary broncho- (catarrhal) pneumonia of childhood. In the production of the lobulated pulmonary consolidation met with in this disease we recognise two factors—the direct extension of the inflammation from the bronchi to the air-vesicles, and the changes produced in the lung as the result of the retained and richly cellular bronchial secretion. Much of this inflammatory secretion becomes drawn in during the respiratory act, and so gives rise to disseminated patches of consolidation, which are merely groups of alveoli filled with the inhaled mucus. This may take place without any marked active changes in the alveolar epithelium, although in other cases the retained secretion produces a considerable amount of epithelial proliferation, and even some cellular infiltration of the alveolar walls.

Another well-known consequence of retained bronchial secretion is the pulmonary collapse and

attendant changes which it may produce. The mucus, by preventing the entrance of air into certain groups of air-vesicles, causes the collapse of these vesicles. The occurrence of the collapse is followed by hyperæmia and œdema of the collapsed portions, and this by active changes in the alveolar epithelium. This often occurs coincidently with the inhalation of mucus, and gives rise to very similar macroscopical appearances. Changes like these, when occurring in previously healthy lungs, tend to terminate in complete recovery. The accumulated alveolar products undergo fatty metamorphosis, and are got rid of by expectoration and absorption, although in some portions of the lung they may remain and form small caseous masses, which ultimately become encapsuled. When, on the other hand, they take place in a lung which is constitutionally weak—one in which there exists a predisposition to phthisis—the result is different. In such a lung, owing to the abnormal susceptibility of the pulmonary tissue to injury, not only is the bronchial implication more severe, but the irritation of the products which have accumulated within the alveoli is sufficient to cause active changes in the alveolar walls; these become thickened by a small-celled growth, and caseation and necrosis may be the ultimate result.

In comparing the pulmonary changes which are

produced by the bronchial secretion in a case of capillary bronchitis in a child with those which constitute phthisis we are, however, met by this difficulty—that the seat of the pulmonary consolidation differs in the two cases. Whereas the simple broncho-pneumonia which results from a capillary bronchitis is most marked in, and often exclusively confined to, the lower portions of the lungs, the consolidation of phthisis almost invariably commences at the apices. The explanation of this difference involves the explanation of the fact that phthisis is *par excellence* an apex disease. On this subject I shall speak presently.

The other way in which catarrh of the smaller bronchi may become a cause of pulmonary consolidation is by its leading to a cellular infiltration of the bronchial walls which gradually invades the adjacent alveoli. We have seen how frequently this occurs in phthisis, where the growth often takes place rapidly, and the consolidation produced quickly degenerates. Even in non-phthisical lungs the much less extensive cellular infiltration of the bronchial walls which results from oft-repeated attacks of bronchial inflammation sometimes extends to and invades the alveoli, so as to produce small tracts of peri-bronchial induration.

Thus far I have considered the effect of injuries inflicted through the medium of the bronchi only

in so far as these have produced bronchial catarrh and infiltration of the bronchial wall. Probably a still more important way in which such injuries produce pulmonary consolidation is by *their direct effect upon the inner surface of the air-vesicles.* When it is remembered that the inner surface of the alveoli is, equally with the bronchial walls, in direct communication with the external air, although the influence of the air must be modified by its passage through the tubes, it will readily be understood that the same injury which affects the bronchi may at the same time affect the alveolar walls. The terminal extremities of the bronchi are, as we have seen, very frequently affected in phthisis, and although possibly the inflammatory process in these may sometimes precede that in the groups of air-vesicles with which they communicate, it would appear to me more probable that the two are, for the most part, simultaneously involved.

Having described how injuries inflicted through the medium of the bronchi may be causes of phthisis, it remains to consider the *nature* of the injury. This is obviously, in most cases, some condition of the respired air. It would be beyond the scope of the present lectures to enter into the various atmospherical conditions which act injuriously upon the respiratory organs. Suffice it to

say, that the one which appears to be injurious above all others is cold and damp. The air may also contain substances which, either from their mechanical or chemical properties, act as direct irritants. The respiration of impure air, and of air containing solid particles, may thus set up inflammatory processes in the lungs. The last-named condition obtains in the case of potters, colliers, and others engaged in similar occupations. In these people the constant respiration of an atmosphere containing solid particles produces that chronic inflammation of the bronchi, and gradual induration and ulceration of the lungs, which is usually known as potters' or colliers', &c., phthisis. This disease, however, usually differs from ordinary chronic phthisis in its greater chronicity, in the textural changes being in the earlier stages peri-bronchial, and in the absence of caseation; although where a phthisical predisposition exists the lungs may exhibit the same characters as those we have been considering—the development of the phthisis being determined and hastened by the irritating influence of the respired air.

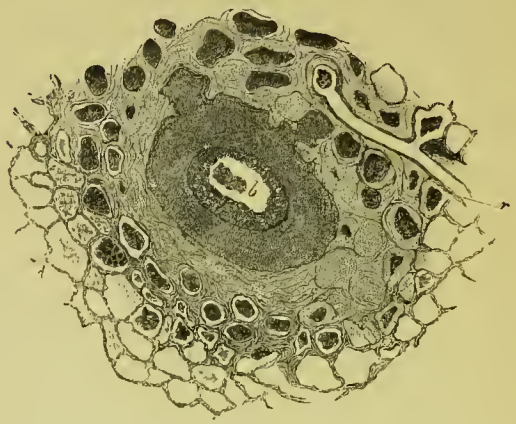
Lastly, I must very briefly allude to the presence of blood in the bronchi as a cause of phthisis, inasmuch as some recent observers have expressed the opinion that bronchial hæmorrhage may give rise to phthisical consolidation of

the lung (hæmorrhagic phthisis). That blood which has been inhaled into the alveoli may, under certain circumstances, set up inflammatory processes in the alveolar walls, and ultimately cause a disintegrative consolidation, is probable. The question is as to whether the hæmorrhage is the sole cause of the phthisis, or whether it is not itself the result of pre-existing phthisical disease. For my own part, I doubt whether blood *per se*, in the absence of a pulmonary predisposition, can cause phthisis; but, given the predisposition, it may perhaps produce sufficient irritation to set up a phthisical process. An accumulation of blood in the lungs in a case of existing phthisis may also tend to cause an increase of the consolidation. As to whether bronchial hæmorrhage occurs independently of pre-existing phthisis, I hesitate to express an opinion. It must certainly be rare, and in many cases it must be difficult to exclude completely the presence of any pre-existing phthisical disease.

In concluding this consideration of the production of phthisis by means of irritation through the medium of the bronchi, it must be remembered that the inflammatory processes which are thus set up are characterised by the disseminated character of their distribution. Groups of air-vesicles communicating with the terminal bronchioles become implicated, and thus are produced lobular lesions

(Fig. 15). These lesions usually ultimately coalesce, and so give rise to more diffused tracts of consolidation. This lobulated distribution of the phthisical consolidation is exceedingly characteristic, and even in those acute cases in which, owing to the rapid and

FIG. 15.



Acute Phthisis. A transverse section of a terminal bronchus (air-passage) and the surrounding alveoli. Showing the lobulated character of the pulmonary consolidation. *b*, cavity of bronchus containing a little mucus. *r*, a blood-vessel. × 50, reduced ½.

extensive implication of the lung, the consolidation may, to the naked eye, appear almost uniform (like a croupous pneumonia), the microscope will usually reveal a lobular character.

The third and last method by which injuries may be inflicted on the lung is by means of *infection*.

By this I mean that irritation of small areas of the pulmonary tissue by means of minute particles derived from some pre-existing inflammatory product, such as occurs in acute miliary tuberculosis. In this general infective disease the source of infection may be situated in any part of the body; but when infection becomes an auxiliary in the production of phthisical consolidation of the lungs, the infective particles are usually derived from some pre-existing phthisical disease. In other words, the infective process usually supervenes upon pre-existing phthisis. Any mass of phthisical consolidation may, under certain circumstances, constitute an infective focus, and so give rise to disseminated inflammatory processes in adjacent or distant tissues, the area of infection depending upon whether the infective materials are distributed by means of the blood-vessels or lymphatics. The infection which is so common in phthisis is that more localised process in which the lymphatic vessels and serous canals are the carriers of the infective agents, so that the secondary inflammatory processes are more or less confined to the neighbourhood of the infective focus. The resulting disseminated lesions are in all respects precisely similar histologically to those which have been already described as occurring in miliary tuberculosis. The part which infection plays in the production of phthisis varies

very considerably in different cases. It is probably not common for phthisical consolidation to exist without, at some time or other, acting more or less injuriously upon the pulmonary tissues. In many cases it causes merely a small amount of secondary consolidation in its immediate vicinity; but in others the infection extends over a much wider area, and may involve the whole of that lung in which the primary disease is situated. It is in this way that infection is sometimes met with as an acute process terminating chronic phthisical disease.

There remains still one other factor which has a most important influence upon the causation of phthisis —*the general health of the individual.* That the development and progress of phthisis is greatly influenced by the state of the general health is well known. That this should be so will be readily understood if the inflammatory nature of the disease be kept in view, inasmuch as, in all cases, the susceptibility of a living tissue to injury is, *cæteris paribus*, greater, and the power of recovering itself from the effect of the inflammation less, the lower the standard of the general health. Consequently, in phthisis the influence both of primary and secondary irritation (infection), the progress of the inflammatory process, and the amount of permanent damage which is inflicted upon the lung, vary according to the health of the individual. And it

must be remembered that, inasmuch as the health of the individual is influenced by the health of the parents, a low standard of health in either parent may so influence the child as to produce a weakness of the tissues generally, in which the lungs participate.

I must now allude to a somewhat difficult subject —why are the apices of the lungs in almost all cases the first to become involved? There must obviously exist some conditions here which are especially favourable to the development of a phthisical process. Much has been advanced during recent years by Aufrecht, Ruehle, Rindfleisch, and others, to show that such conditions are connected with imperfect respiratory power. This question has been ably discussed by Professor Ruehle in his article on Pulmonary Consumption in Ziemssen's "Cyclopædia of Medicine,"* and I will here only briefly indicate some of its more important bearings. That the range of respiratory movement of the pulmonary apices is, in many individuals, somewhat less than that of the middle and inferior portions of the lungs is, I presume, pretty generally admitted. An interference with

* See the article on Pulmonary Consumption in Ziemssen's "*Cyclopædia of the Practice of Medicine*," vol. v. p. 486.

the respiratory power of the highest portions of the lungs, for example, is often caused by a faulty carriage of the body. The stooping posture, the shoulders being thrown forward so that the weight of the arms falls on the thorax, must materially restrain the movements in the clavicular and subclavicular regions. In that diminution of the respiratory capacity of the lungs which obtains in so many of those who become the subjects of phthisis, and which, according to Ruehle, appears to be mainly due to weakness of the thoracic muscles, it is in the highest portions of these organs that the deficient expansion is the most marked. Then, again, as pointed out by this observer,* the situation of the apex of the lung above the clavicle and outside the chest, must tend to interfere with its movements in respiration.

If it be admitted that the respiratory movements of the upper portions of the lungs may thus be impaired, the favourable influence of such impaired movement upon the development of a phthisical process is obvious. Diminished respiratory power of the apex of the lung must tend, in the first place, to interfere with the removal by expectoration of any inflammatory products which may exist in the bronchi or air-vesicles in this situation; and

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* *Loc. cit.*

any accumulation of such products would obviously favour the development of phthisis. This difficulty of removing by expectoration secretions from the upper portions of the lungs I think we meet with not unfrequently in cases of simple bronchial catarrh, occurring in debilitated subjects, or in those enfeebled from acute disease. In these the râles sometimes persist at the apices for some time after they have disappeared from other parts. Another result of impaired respiratory power must be to cause some slowing of the blood-stream in the pulmonary capillaries; and the passive hyperæmia thus induced will not only favour bronchial and alveolar catarrh, but also hinder the removal by absorption of any accumulated inflammatory products. Although other circumstances are probably concerned—and our knowledge on the subject is undoubtedly most incomplete—I am inclined to believe, with Professor Ruehle, that it is to some of these results of imperfect respiratory power that the commencement of phthisical processes in the apices of the lungs is in great measure to be ascribed.

Having now considered somewhat fully the histological changes met with in the lungs in phthisis, and the nature and etiology of the morbid processes upon which they depend, the question arises as to how far a subdivision of the disease into different *pathological* varieties is admissible. In

attempting to answer this question, I would again state that I do not wish to be understood to include within the category of phthisis all forms of chronic lung-consolidation which are attended by cough, expectoration, and emaciation. We must admit the existence of a chronic pneumonia, leading to pulmonary fibrosis, bronchiectasis, and secondary ulceration of the indurated lung; of ulcerative destruction of the lung due to obstructed bronchus; and of pulmonary syphilis. If these be regarded as phthisical lesions they undoubtedly represent distinct pathological conditions; but for reasons I have already given they are excluded from our present inquiry. Phthisis thus limited in its signification will, I presume, include those varieties often described as pneumonic, tuberculo-pneumonic, tubercular, and scrofulous phthisis, and also many fibroid forms of lung disease. Are there any grounds for making such pathological distinctions as these terms represent? All phthisis is inflammatory, and I cannot but think that to apply the term "pneumonic" to those cases in which the process happens to be of considerable intensity tends to mislead. With regard to the terms "tuberculous" and "scrofulous," as applied to phthisis, tuberculous, if it means anything, means, I think, that cellular infiltration of the alveolar and bronchial walls which gradually extends and invades the

lung, producing varying-sized tracts of bloodless, caseating consolidation. "Scrofulous" has obviously a precisely similar signification. This cellular infiltration, as we have seen, is invariably present, although its extent varies very considerably in different cases; and those cases in which it is the most pronounced may, if you will, be termed scrofulous or tuberculous phthisis. The term "fibroid" is applicable to those cases in which pulmonary fibrosis is the prominent lesion. Fibrosis tends to become marked in all cases which are of great chronicity; and in these it may sometimes almost completely mask the lesions which occurred in the earlier stages of the disease. From a careful consideration of the histology and pathology of phthisis, I am therefore inclined to doubt the advisability of attempting, as has been done during recent years, to subdivide the disease into distinct pathological varieties. The variations met with in the clinical history of phthisis and in the physical characters of phthisical lungs are, I think, mainly to be ascribed to variations in the intensity and duration of the inflammatory processes which give rise to the pulmonary consolidation, and also to the parts which primary pulmonary inflammation, bronchial inflammation, and infection play respectively in the causation of the disease.

I would here say one word on the subject of phthisis and syphilis. Is there a syphilitic phthisis? There is, as I have already stated, a disease of the lung which is distinctly syphilitic, but it is, I believe, quite rare; it usually affects some other portion of the lung than the apex, and it differs in many other respects both histologically and clinically from ordinary phthisis. Excluding, however, these rare cases of syphilitic lung disease, we must admit that phthisis is often materially modified by the existence of syphilis. Syphilis, in the first place, by injuring the general health, must predispose to phthisis. We have probably most of us met with cases of chronic phthisis in an early stage, occurring in those who are the subjects of marked syphilis—cases which presented nothing unusual either in symptoms or physical signs, but in which marked improvement has followed the use of anti-syphilitic remedies. Such cases, I presume, cannot be regarded as examples of a syphilitic variety of phthisis. They present, as far as I know, nothing histologically distinctive, and it must be borne in mind that in those who are the subjects of syphilis, many diseased conditions which present in themselves nothing specific, are favourably influenced by treatment which has for its object the destruction of the syphilitic poison.

In concluding these lectures on the pathology of phthisis, it may be asked what bearing such pathological questions have upon the prevention and treatment of the disease. To this I would reply, in the first place, that the older doctrines which were held respecting the specificity of tubercle and phthisis undoubtedly exercised an unfavourable influence upon treatment. During recent years there has been a gradually increasing tendency to regard phthisis as an inflammatory affection, and there has consequently been a corresponding tendency to attempt by treatment to prevent the occurrence and control the influence of bronchial and pulmonary inflammation. Continued pathological research points, I think, to the necessity of moving still further in this direction. We do not perhaps even yet sufficiently recognise the fact that the development of phthisis is determined, and the progress of the disease influenced, by the ordinary causes of inflammation; and the results of pathological investigation indicate, I think, the advisability of directing our treatment still more closely with the object of preventing and rapidly controlling all inflammatory processes in the lungs. Of all the teachings of our pathology this is undoubtedly by far the most important.

The influence of the general health upon the

development and progress of phthisis, and the importance of maintaining and improving it, is so fully recognised that it is scarcely necessary to allude to it. At the same time, it is interesting to note how completely this influence is accounted for by the inflammatory nature of the disease. Not only is the susceptibility of the pulmonary tissues to injury greater, and the power of recovering from the inflammation which is induced less, the lower the standard of the general health; but, as we have seen, the state of health materially influences the tissue-changes which accompany the inflammatory process. It is in the scrofulous and in those whose constitutions are otherwise impaired that pulmonary inflammations tend to be protracted and obstinate, thus leading to that cellular infiltration of the alveolar and bronchial walls which constitutes the most important histological factor of phthisical disease. By maintaining and improving the general health we shall best lessen the susceptibility of the lungs, which are too often constitutionally feeble, to the various injurious influences to which they are exposed, and thus do very much towards preventing the development of phthisis. By the same means also, where the disease is already established, we shall help to arrest its progress; and the improved health will beneficially influence

its course, not only in the way I have just indicated, but also by diminishing the results of that injurious (infective) influence which is so often exercised by any existing phthisical consolidation of the lung upon neighbouring, and sometimes upon more distant, parts.

I would now say a few words on the subject of apex disease. What indications are afforded by pathology for the counteraction of that special liability to phthisical processes which exists in the upper portions of the lungs? Although our knowledge of the causes of this liability are still very incomplete, we have seen that there is good reason to believe that imperfect respiratory movement is an important causative factor. Such being the case, treatment calculated to counteract those various conditions, either inherited or acquired, which tend to impair the expansion of the pulmonary apices, ought to be beneficial. That this is the case as far as the prevention of phthisis is concerned will, I presume, be pretty generally admitted. The value of calisthenic exercises and of other measures which promote the development of the respiratory muscles and the full expansion of the chest, as an auxiliary to treatment which has for its object the prevention of phthisis, can, I think, scarcely be questioned.

But although such measures may be of use as tending to diminish the liability to phthisis, the question arises as to how far they are indicated when the disease is already established. On this subject we must, I think, speak with great caution. When active changes are taking place in the lung, means calculated to restrain the respiratory movements are probably more likely to be beneficial than any which would increase them. With this object, a treatment of phthisis by strapping, and other mechanical appliances which limit the movements of the upper parts of the chest, has lately been employed. Whether such treatment is ever useful a lengthened experience alone will show, but it would appear to me that it is only likely to be so in some few cases—perhaps more especially in quite the early stage of certain acute and localised processes. On the other hand, inasmuch as by restricting the respiratory movements we tend to diminish the activity of the pulmonary circulation, and thus to interfere with the absorption of inflammatory products, such treatment would appear to be contra-indicated in cases where the disease is not active, and where perhaps much intra-alveolar matter exists which is capable of removal. In these last-named cases attempts to stimulate the circulation in the diseased lungs by cold douches,

friction, and other similar means, such as has been recently advocated by Dr. A. Von Sokolowski, would appear to me to be more in accordance with the teaching of pathology.*

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* "Berliner Klinische Wochenschrift," Nos. 39 etc. 1876.

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PRINTED BY BALLANTYNE AND HANSON
LONDON AND EDINBURGH

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